DISSERTATION ON

A CLINICAL STUDY ON CYSTOID MACULAR EDEMA DUE TO VEIN OCCLUSION AND ITS MANAGEMENT

Submitted in partial fulfillment of requirements of

M.S.OPHTHALMOLOGY
BRANCH – III

REGIONAL INSTITUTE OF OPHTHALMOLOGY
MADRAS MEDICAL COLLEGE
CHENNAI – 600 008

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVESITY

CHENNAI

APRIL 2017

CERTIFICATE

This is to certify that the dissertation titled, **"A CLINICALSTUDY ON CYSTOID MACULAR EDEMA DUE TO VEIN OCCLUSION AND ITS MANAGEMENT** is a bonafide record ofthe research work done by DR.SHALINI. S, post graduate in the RegionalInstitute of Ophthalmology & Government Ophthalmic Hospital, MadrasMedical College and Research Institute,Chennai-08,submitted in partialfulfillment of the regulations laid down by the Tamil Nadu Dr. M.G.R.Medical University, Chennai for the award of M.S.OphthalmologyBranch III, under my guidance and supervision during the academic years2014 - 2017

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I hereby declare this dissertation entitled **"A CLINICAL STUDYON CYSTOID MACULAR EDEMADUE TO VEIN OCCLUSION AND ITS MANAGEMENT** is a bonafide and genuineresearch work carried out by me under the guidance ofProf.Dr. M RajakumariM.S , D.O

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ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to**Prof.Dr.M K Muralidharan. MS MCH,** Dean, Madras Medical College,for permitting me to conduct this study.
I have great pleasure in thanking **Prof. Dr.WaheedaNazir M.S., D.O.,** Director and Superintendent, RIO-GOH,Madras Medical College,Chennai, for her valuable support.

I have great pleasure in thanking **Prof.Dr M .Rajakumari M.S.,D.O.,** Head of Department of Uvea Retina services, RIOGOH, andguides in this study for their valuable guidance and constant support atevery stage throughout the period of this study.

I express my sincere thanks to **Prof. Dr.K.Namithabhuvaneshwari MS,DO,** former Director of the RIOGOH and **Prof V Revathy MS,DO** former chief of uvea retina services and currently the project director of RIO –GOH for guiding me in my initial stages at this institute and instilling an interest in the field of Ophthalmology.

I am grateful to my unit Assistant Professors, **Dr. K. RavikumarM.S., Dr.A. Nandhini M.S. , Dr P. Shoba M.S., Dr.M. Periyanayagi M.S.,** for their constantsupport and guidance throughout my period of study at this Institute.

They have been responsible for all that I have learnt during this period.Their suggestions were invaluable additions to this study.I am also indebted to all Professors and Assistant professors of thisInstitute for the help and guidance rendered during my period of study atthis Institute. I also place on record my

thanks and appreciation of thework and support received from all my colleagues during my studyperiod.

I am greatly indebted to all my patients for their kind consent andco-operation which made this study possible and without which I wouldnot be the person I am.“Thanks” would be too small a word for my mother, father and my sister for their countless and unconditional sacrifices over the years. My deepestgratitude and love goes to them for their unflagging love and support.I am ever grateful to the almighty for always showering hisblessings on me.

ABSTRACT

TITLE - "A CLINICAL STUDY ON CYSTOID MACULAR EDEMA DUE TO VEIN OCCLUSION AND ITS MANAGEMENT "

AIM - To compare the functional , anatomical and therapeutic outcome of patients with cystoid macular oedema due to retinal vein occlusion .

MATERIALS AND METHODS – This iaa prospective study conducted at Uvea and Retina department, RIOGOH, Egmore,. About 30 patients were analysed and followed upto 3months on regular basis and as and when required.

OBSERVATIONS AND RESULTS : The mean age of presentationwas 53.2 ± 5.6 years with a male : female ratio of 1.9:1. Supero temporalbrvo was found to be the leading cause. The reduction in mean macular thickness was significant in patients .

After treatment all patients had a significant 2 Snellen line visualimprovement. The correlation between macular thickness and visualacuity was weak positive in post treatment. There was no Increase in intraocular pressure noted. Hypertension was considered as a major association in developing vein occlusions.

CONCLUSION : The fall in Mean macular thickness after IVTA and Intra vitrealAntiVEGFwas statistically significant in all patients analysed.The visual

acuity improvement also was statistically significant ($p<0.0001$) butcorrelation with macular thickness was weak positive. Intravitrealtriamcinolone acetonide is a promising therapy in refractory cystoidmacular edema in terms of safety and effective.

KEYWORDS -Cystoid Macular Edema, Central Macular Thickness, VeinOcclusion, Visual Acuity.

CONTENTS
PART – I

PART- II

PART- III

ABBREVIATIONS

CME – Cystoid macular edema

FFA – Fundus fluorescein angiography

OCT – Optical coherence tomography

AC – Anterior chamber

NSAIDs – Non steroidal anti inflammatory drugs

Nd-Yag – Neodymium Yttrium Argon Garnet

ILM – Internal limiting membrane

CNVM – Choroidalneovascular membrane

CSR – Central serous retinopathy

RPE – Retinal pigment epithelium

NPDR – Non proliferative diabetic retinopathy

FAZ – Foveal avascular zone

CRVO – Central retinal vein occlusion

BRVO – Branch retinal vein occlusion

ERM – Epiretinal membrane

DM – Diabetes mellitus

TNF – Tumour necrosis factor

CSME – Clinically significant macular edema

ACIOL – Anterior chamber intraocular lens

PCIOL – Posterior chamber intraocular lensOPL – Outer plexiform layer

DME – Diabetic macular edema

NFL – Nerve fibre layer

PAM – Potential acuity meter

ERG – Electroretinogram

FERG – Fovealelectroretinogram

ETDRS – Early treatment Diabetic Retinopathy study

FDA – Food and drug administration

RP – Retinitis pigmentosa

PPV – Parsplanavitrectomy

ARMD – Age related macular degeneration

VEGF – Vascular endothelial growth factor

PKP – Penetrating keratoplasty

IVTA – Intravitreal triamcinolone acetonide

SCORE – Standard vs Corticosteroid for retinal vein occlusion study

RD – Retinal detachment

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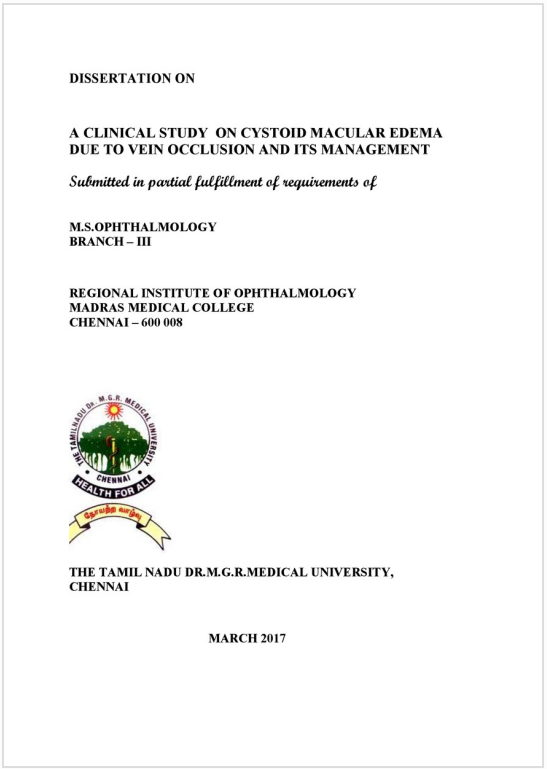

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MARCH 2017

INTRODUCTION

Retinal edema is most commonly seen due to circulatorydisturbances and inflammatory conditions affecting the eye. Macular edema is broadly defined as an abnormal thickening of macula caused by excess accumulation of fluid in the extracellular spaces of retina.

Cystoid macular edema is condition caused by excess accumulation of fluid in cyst likespaces within the maculamostly in the outer plexiform layer. It is a commonpathological response to a wide variety of ocular insults. It is thought thatprostaglandin secretion and vascular endothelial damage causes fluidaccumulation in the relatively loose intercellular adhesions of outerplexiform layer which permits the formation of cystoid spaces.

Macular edemais the most important cause for loss of central vision in most patients.

Retinal vein obstructions is another common cause of CME. In patients with central retinal vein occlusion or a tributary branch occlusion and if macula is involved CME is a major cause of visual loss. ifedema is massive or chronic (8 months), it causes permanent diminution of vision

due to disruption of the intraretinal connections and to the intracellular damage suffered by the visual elements . Persistent CME may be associated with vitreomacular attachment or hyperlipidemia and cardiovascular disease. Finkelstein proposed a theory saying ischemic CME following branch retinal vein occlusion is often transient and, compared to perfused CME, has better prognosis for visual acuity. Another important sign of CME following venous obstruction is the development of fluid blood levels in central cystoid spaces which is more common in venous occlusion compared to other pathology.

Aphakic and Pseudophakic cystoid macular edema, commonlycalled as Irvine-Gass syndrome, has been recognised as a distinctentity since 1953 after its description by Irvine and Gass and by Norton in1966. It is one of the most frequent and troublesome problem followingcataract surgery. With recent improvements in suture material production instrumentation , newer techniques and antibiotic therapy, loss of central vision secondary to changes in macula following uneventful cataract extraction has received recognition as a major complication of cataract surgery.

REVIEW OF LITERATURE

HISTORY

1877 - Thevery first case of branch retinal vein occlusion was reported byLeber.

1899 – Retinal vein occlusion is seen more commonly on the temporal than the nasal part of retina by Ammann

1928 – Koyanagi first reported association of vein occlusion with arterio – venous crossing and these causing macular edema.

1953 – Irvine reported a syndrome of spontaneous rupture of thehyaloid face following uneventful cataract extraction. There was decreased visual acuity secondary to vitreous opacities and macular degeneration along with irritability of the eye affected.

1954 – Schepens noted attachment of the vitreous at the vitreousbase, disc and macula .

1955 - Schepens reported macular clouding followed by a macular break which in turn lead to retinal detachment, due to vitreous shrinkageand traction.

1965 – Tolentino and Schepens presented a series of cases of retinal edemaafter cataract extraction. In most of them they were able to demonstrate vitreous strands attaching to the macula and occasionally to optic disc. They attributed the change to vitreo-retinal traction.

1966 – Gass and Norton framed the title “Cystoid Macular Edema”. They illustrated the characteristicclinical and FFA picture and described in detail the biomicroscopicappearance of the condition

1966 – Iliff gave the vitreous traction theory.

1968 – Gass and Norton did partial open sky vitrectomy undermicroscopic control using cellulose sponge and scissors.

1970 – Mechanized vitrector was used for limbal and parsplanavitrectomy.

1977 – Miyake first used NSAIDs like topical indomethacin in thetreatment of CME following cataract surgery.

1983 – Katzen, Fleischman, Trokel used the Nd-Yag laser forvitreolysis.

1991 – Shahidi, Ogura determined that retinal thickness asmeasured by biomicroscopy and stereophotography correlates well withvisual acuity.

1995 – Arend and Remby used fluorescein angiography generatedwith scanning laser ophthalmoscope for early recognition of cystoidformation in CME.

1995 – Hee and Puliafito made use of optical coherence tomography for objective monitoring of retinal thickness in patients with CME

ANATOMY OF MACULA

The **macula** or **macula lutea** (from Latin *macula*, "spot" + *lutea*, "yellow") is an oval-shaped pigmented area in thecenter of theretina . There is no anatomical landmark to define this zone on clinical examination or on morphological basis. It is approximately a circle with radius of 2.75mm centred at fovea (5.5mm in diameter). The yellowcolour of macula is due to xanthophyll pigments in ganglion cells. The

side walls of macula slope gently towards the fovea centralis.

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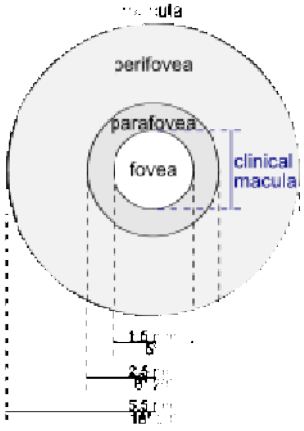
Fovea centralis is the smalldepression in the inner retinal surface in the centre of macula and measures 1.5mm in diameter and 0.25mm in thickness It is a small pit that contains the largest number of cone cells. Foveola is 0.35mm in diameter and 0.13 mm in thicknessoccupying centre of fovea. It is situated 4mm temporal and 0.8mminferior to optic nerve head. Rod cone ratio is about 1:2 in this region.A small depression in the centre of foveola is called umbo.

Parafoveal zone is an area measuring 0.5mm surrounding thefovea. Rod cone ratio is about 1:1.

Perifoveal zone is 1.5mm wide zone surrounding the parafovealarea.



BLOOD SUPPLY OF MACULA

Macular region is supplied by superior and inferior branches of central retinal artery. In 20% of individuals, cilioretinalartery also supply the macula.

Capillaries are arranged as three layered in the macula and they arereduced to single layer in the perifoveal area and in centre is the capillary

free zone of 400-600μm in diameter.

BLOOD RETINAL BARRIER

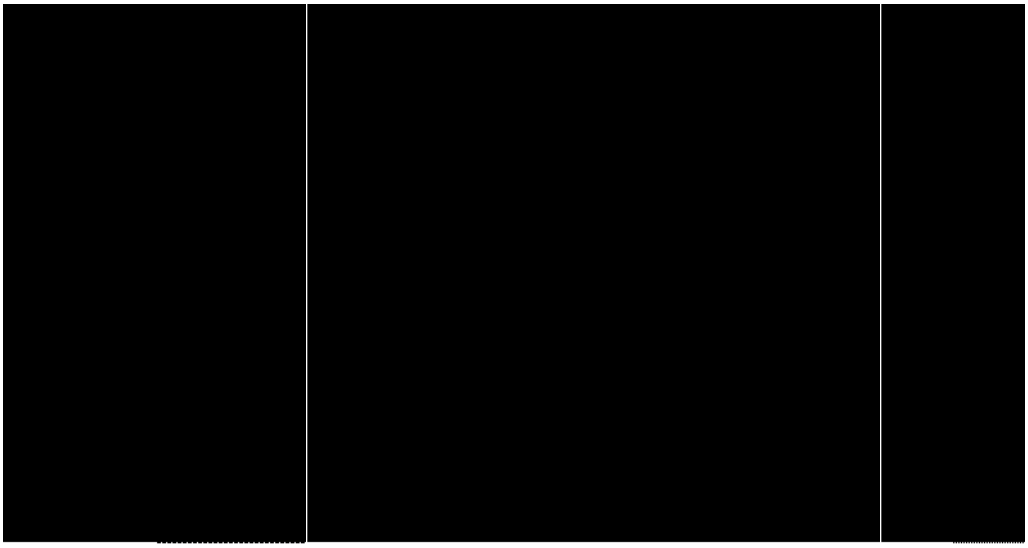
Outer Blood Retinal Barrier

This is formed by the tight junctions (Zonulaeoccludens andZonulaeadherens) of retinal pigment epithelial cells. Helps to regulate the movement of solutes and nutrients from the choroid to the sub-retinal space

Inner Blood Retinal Barrier

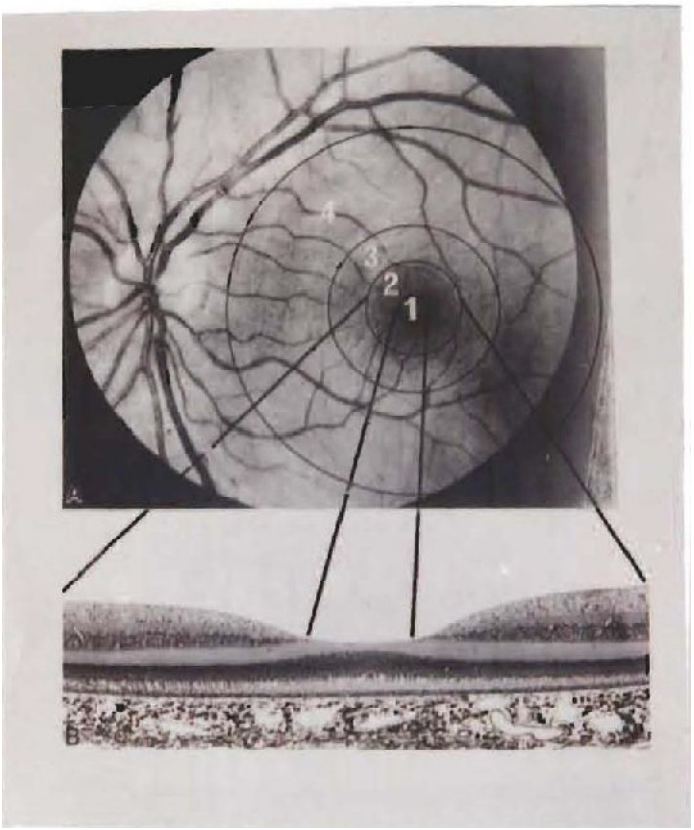
The endothelial cells of retinal capillaries are bound together aboutthe lumen by intercellular junctions of zonulaeoccludens type and forms

the inner blood retinal barrier.



Schematic diagram of the blood-ocular barrier

ANATOMY OF FOVEA



(1) Foveola (2) Fovea centralis (3) Parafovea (4) Perifovea

MICROANATOMY OF MACULA

Retina at macula is made of three types of cells and their synapsesarranged from without inwards in the following layers

- Retinal pigment epithelium
- Layer of rod and cones
- External limiting membrane
- Outer nuclear layer
- Outer plexiform layer
- Inner nuclear layer
- Inner plexiform layer
- Ganglion cell layer (multilayered in comparison to rest of retina)
- Nerve fibre layer
- Internal limiting membrane

In Cystoid Macular Edema there is accumulation of fluid betweenouter plexiform and inner nuclear layer of the retina.

FOVEA CENTRALIS

This region is predominated by cones and are arranged obliquelyforming the Henle's layer

FOVEOLA

This region of retina contains cones and their nuclei covered bythin internal limiting membrane. Remaining retinal layers (Inner nuclearlayer, Inner plexiform layer, ganglion cell layer and nerve fibre layer) areabsent in foveola.

HENLE'S FIBRELAYER

The outer plexiformlayer of retina is made up of arborisation of axons of rods and cones with bipolar cell dendrites. It includes Muller's fibres and Horizontal cell processes. This layer has a reticular structure but asmacula is approached it takes up a fibrous structure called Henle's layer.

The fibres run vertically at first and then obliquely near macula andfinally parallel to surface which is thickest at macula and absent atfoveola. There is progressive disappearance of rods.(1)

ANATOMICAL PECULIARITIES OF MACULA CAUSING AN EXAGGERATED RESPONSE TO PATHOLOGICAL PROCESSES

The peculiar susceptibility of the macula to a number of differentpathological processes, both local and general is called the "exaggerated

response of macula" (2). Anatomical causes for this are:

Vascular supply – The arcade arrangement of capillaries whicharise as terminal parts of an end artery system together with the centralavascular zone make the foveolar region a watershed. Local impairmentof metabolism whether from disturbances of perfusion and accumulation of metabolites or from the effects of capillary damage leads toextracellular fluid collection at a quicker rate than they can be absorbed. Also the capillaries at fovea are longer and vascular complexes arethinner than elsewhere hence they are more susceptible to noxioussubstances.

1. Tissue architecture – The processes of Muller's cells runhorizontally in outer plexiform layer hence retina loses its compactnature and this laxity causes large amount of extracellular fluidto be accumulated in the macular region leading to characteristiccystoid edema

2. Cellular constituents – The ganglion cells have high metabolicactivity and their dysfunction leads to rapid accumulation of tissuemetabolites. Most of these have a vasodilator effect and this alongwith the underlying hypoxia leads to edema.

3. Internal limiting membrane – The vitreous is an excellent diffusion

medium and the ILM provides little additional interference to theprogress of toxic substances diffusing across it. Inflammatorytoxins arising from the iris, peripheral choroid and pars plana maytraverse the vitreous and because of the thinness and adherence ofILM in foveal region, which may disturb the function of cells which are highly concentrated around foveal rim and also affect the capillary permeability of the macular region

4. Choroid and RPE – The macular choroid and RPE are also the most important sites for degenerative changes which may behereditary, toxic or arteriosclerotic in nature. There is apredisposition for choroidal vascular disease with decompensationand hemorrhage in the central area.

EVALUATION OF MACULAR DISEASES

SLIT LAMP BIOMICROSCOPY- Utilises high power convex lenses to obtain wide field of view of the fundus which is vertically inverted and laterally reversed and provides high magnification with stereopsis to detect macular disease.

AMSLER GRID TEST - evaluates central 200 of visual field on fixation and henceuseful in screening and monitoring the macular disease. There are 7charts. Chart 1 is most commonly used. This chart consists of white gridon black background with 400 small, 5mm squares, each square

subtendsan angle of 10 when viewed at 33cm. Each eye is checked individually,with the chart held at 33cm with prior correction for presbyopia. Patientsare asked to maintain fixation on the central dot and comment on fourcorners, any missing areas on chart and wavy lines.

FUNDUS FLUORESCEIN ANGIOGRAPHY - Fluorescence is the property of certain molecules to absorb light of shorter wavelength and emit light of longer wavelength. S odium fluorescein is a water soluble orange dye, 3ml of 25% fluorescein is injected intravenously through antecubital vein, 85% isbound to plasma proteins and remains intravascular. Passage of dyethrough retinal and choroidal circulations is studied through photographicsurveillance.

Phases in FFA – Choroidal phase, Arterial Phase, Arteriovenousphase, Venous phase, Recirculation phase

Causes of Hyperfluorescence – Autofluorescence, Pseudofluorescence, Window defect, Pooling, Leakage, Staining.

Causes of Hypofluorescence – Masking of Retinal fluorescence,Masking of Choroidal fluorescence, Filling defects.

OPTICAL COHERENCE TOMOGRAPHY

OCT is a non invasive, non contact imaging system that provideshigh resolution cross sectional images of retina, optic nerve head and thevitreous[5]

OCT is analogous to B-scan ultrasonography but uses near-infrared light interferometry rather than sound waves.

OCT is used to differentiate lamellar and full thickness macularhole, to determine treatment options in CNVM, monitoring the course ofCSR, retinal thickness map, to identify type of macular edema and tomonitor its course and so on.

- High Reflectivity – Nerve fibre layer, RPE, Choriocapillaries, Pigment accumulation, Naevus, Neovascularisation, RPE hypertrophy.
- Medium reflectivity – Plexiform layer.
- Low Reflectivity – Nuclear layer, Photoreceptors, Retinal edema,

Cystoid edema, Cavity, Cyst, Pigment epithelial detachment, Serous retinal detachment

The macular edema patterns seen on OCT are as follow:

1. Sponge like Retina - It is mostly confined to outer retinal layers due to backscattering from intra retinal fluid.

2. Cystoid macular edema

Cystoid spaces confined to outer retina mostly. In long standingcases they fuse to form large cyst.

3. Serous retinal detachment

Hypo reflective space under fovea. It may disappear followinglaser.

4. Tractional retinal detachment

Foveovitreal traction causes detachment of fovea. Its an indicationfor Pars Plana Vitrectomy to release traction. Laser will worsen macularedema in these cases.

5. Taut Posterior Hyaloid Membrane

Cystoid spaces confined to outer retina mostly. In long standingcases they fuse to form large cyst.

CYSTOID MACULAR EDEMA

The extracellular space of the retina usually constitutes a smallproportion of its total volume[4] Active transport of electrolytes and largermolecules from retina across the retinal pigment epithelium occurs in this space. Disruption of either inner or outer retinal barrier leads to leakage of plasma proteins and water which leads to expansion of extracellular space of retina. This is often accompanied by accumulation of fluid in the outer

plexiform and inner nuclear layer of retina[6]. Retinal edema localized to macula is called macular edema leading to diffuse thickening of posterior pole. Accumulation of fluid in cystic spaces leads to cystoid macular edema.[1,2]

CAUSES OF CYSTOID MACULAR EDEMA

Cause of cystoid degeneration of macula as described by DukeElder(3) are

1. Senile degeneration.

2. Vascular disorders - Arteriosclerosis, CRVO, CRAO, Retinalperiphlebitis, Hypertensive retinopathy, Diabetic retinopathy.

3. Inflammatory conditions - chorioretinitis, iridocyclitis.

4. Degenerative conditions of macula - Retinal dystrophies, Retinitis pigmentosa.

5 Trauma - usually in contusions or associated with retention of foreign body, radiation injury like eclipse blindness.

6. Glaucoma.

7. Hereditary macular dystrophy.

Other conditions as mentioned by Stephen J. Ryan are(4)

8. Drugs - Epinephrine maculopathy, Nicotinic acid maculopathy.

9. Post surgical - After any type of cataract surgery, vitrectomy,glaucoma procedures, penetrating keratoplasty.

10. Tumours - choroidalhemangioma, choroidal melanoma.

11. Retinal detachment.

12. Tractionalmaculopathies – Epiretinal membrane,Vitreomacular traction syndrome.

13. Optic nerve head pathologies – Optic disc pit, coloboma,diabetic papillopathy.

Retinal Vein Occlusion Induced Cystoid Macular Edema

In CRVO and BRVO, macular edema tends to be chronic, difficultto treat and causes a gross loss in visual acuity. Visual loss is exacerbated by macular haemorrhages, ischemia, secondary RPE changes like ERM.

Pathophysiology Elevation in intravascular pressure in the veins distal to occlusion site causes increase in transmural hydrostatic pressure in retinal capillaries causing a greater transudation of fluid into extracellular spaces.[7]

- Disruption of microscopic intraretinal connections due to cytokines mediated by hypoxia in retina greatly facilitates extravasation of fluids as well as large protein molecule(5).

- Development of fluid blood levels in central cystoid spaces is characteristic of obstructive retinopathy.

Branch Retinal Vein Occlusion

Branch retinal vein occlusion, occurs at an arteriovenous (AV) crossing is the most common RVO. The artery and vein have a common adventitial sheath at the AV crossing with varying degrees of fusion of the vascular wall. Venous compression by the relatively sclerotic artery may result in turbulent flow, endothelial damage, thrombosis and occlusion. [1] Most BRVO occur in the superotemporal quadrant, due to the increased number of arteriovenous crossings in this quadrant. Because of their asymptomatic nature, nasal occlusions are less likely to be diagnosed .[8]

CLINICAL FEATURES

- Complaints of decreased vision or may be asymptomatic.
- Vision loss at presentation is related to the extent of macular damagefrom intraretinaledema,hemorrhage or capillary non-perfusion.
- In the acute presentationthe involved retina typically shows venous dilation and tortuosity, superficial intraretinal hemorrhages, and/or cotton wool spots.

FFA

Findings include delayed venous filling in the area of occlusion, capillary non-perfusion, and macular edema in the early phase , as well as microvascular abnormalities in later stages.Long-term sequelae include sheathing of veins, vascular telangiectasias, the formation of collateral retinal vessels,

Fig 1 – delayed filling in the venous phase and development of collaterals. Few capillary non perfusion areas are seen .

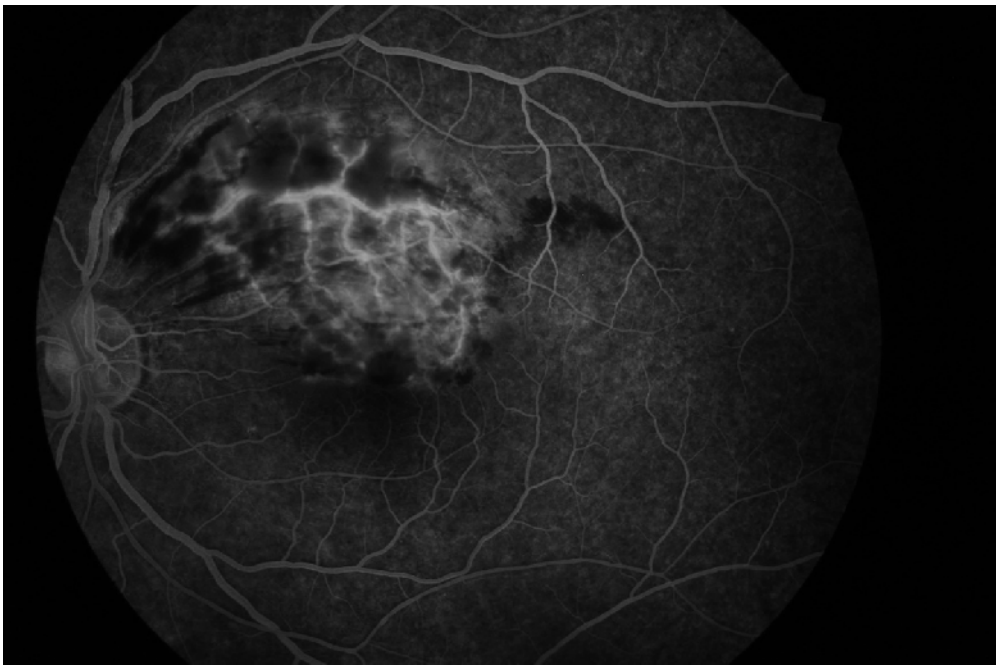


Fig 2 – delayed filling in the venous phase along with blocked fluorescence suggestive of hemorrhages

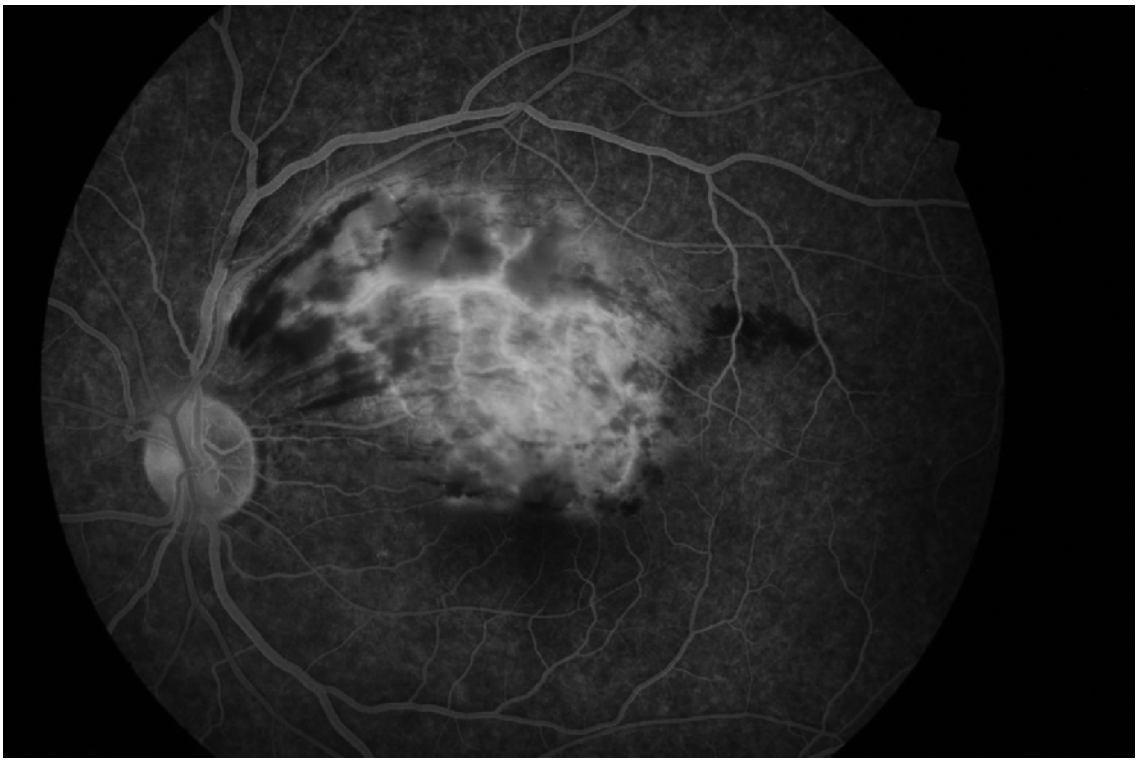
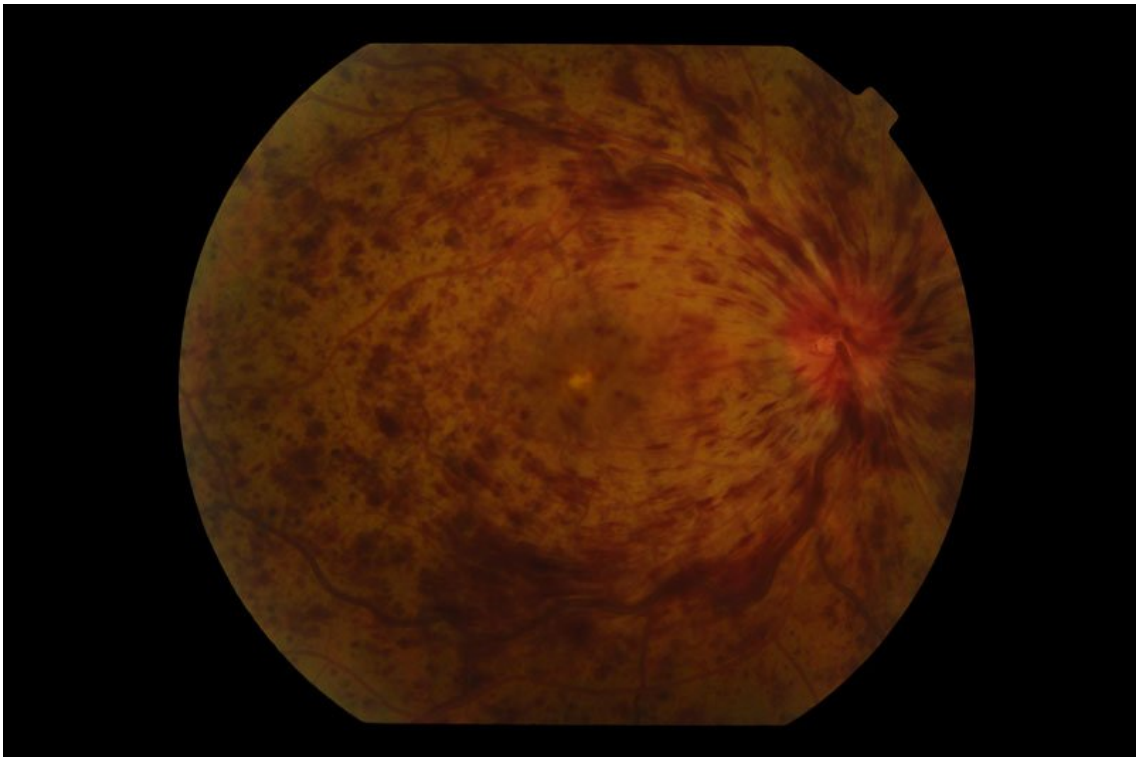


Fig 3 -delayed filling in the early phase with leaks.

OCTshows serous macular detachment and subretinal haemorrhage along with macular edema , cystoid in nature due to accumulation of the fluid .

Central Retinal Vein Occlusion presents with haemorrhage in all 4 quadrants showing a tomato splashed appearance[9]. The occlusion typically occurs at or near the lamina cribrosa, where relative narrowing of the central retinal artery and vein contributes to turbulent flow and an increased risk of thrombosis, CRVO is classified as ischemic and non-ischemic



CLINICAL PRESENTATION

Clinically, patients with CRVO present with sudden loss of central vision.. In the acute phase all four quadrants shows features of venous

dilation and tortuosity, superficial and deep intraretinalhemorrhages, and/or cotton wool spots. There is usually disc edema and there may be macular edema and/or serous detachment. In CRVO, vision loss is primarily due to macular edema, macular hemorrhage, and perifoveal capillary non-perfusion in the ischemic variety[10]. Clinically, visual acuity <20/200, the presence of an afferent papillary defect, and extensive retinal hemorrhages are consistent with ischemic CRVO.

Although the severity of the ophthalmoscopic findings varies with the extent of retinal ischemia, ophthalmoscopy alone is not adequate to differentiate between ischemic and non-ischemic CRVO.

Hemi-Retinal Vein Occlusion:

Hemi-retinal vein occlusions are variants of central retinal vein occlusions that involve the superior or inferior half of the retina.[12]This is due to an anatomic variation at the optic nerve head

OTHER CAUSES OF CYSTOID MACULAR EDEMA

Diabetic Cystoid Macular Edema

It is the most common cause of visual loss in patients with NPDR. This is best detected by slitlamp biomicroscopy. Incidence of macular edemaincreases with type of diabetes, duration of diabetes, age of onset, use of insulin, uncontrolled diabetes, associated risk factors like hypertension, hyperlipidemia, anemia,and nephropathy. It is more common in type 2 diabetes.[11]

Pathophysiology

Hyperglycemia is the main inciting factor which causes disruptionof retinal vasculature that breaks blood retinal barrier and leakage

frommicroaneurysms and capillaries. Focal retinal hypoxia causes an increase

in hydrostatic pressure that in turn increases luminal hydrostatic pressure.This causes dilatation of capillaries with disruption of tight junctionsbetween endothelial cells and favouring fluid egress and macular edema.

Clinical features Of Diabetic macular edema

1. Thickening of macula

2. Blurring of underlying choroidal vascular pattern

3. Loss of foveolar reflex

4. Cystoid spaces

5. Lipid exudation from leaking microaneurysms forming circinate maculopathy.

Classification of type

1. Focal Edema – Areas of focal leakage from microaneurysms forming partial or complete ring of hard exudates delineated from adjacent healthy retina.

2. Diffuse Edema – Areas of leakage from microaneurysms and dilated capillary segments throughout posterior retina causes diffuse edema. It differs from focal edema by

a)Diffuse edema is not associated with hard exudates.

b) It develops cystoid spaces more commonly and better seen in late phases of FFA and on OCT as hyper reflective spaces with hyper reflective septa.

c) It is bilaterally symmetrical.

d) It may disappear spontaneously even without laser only to reappear spontaneously.

e) Systemic features may be associated with exacerbations and amelioration of diabetic macular edema.

3. Ischemic Edema – It is associated with enlargement of FAZ, irregularties of FAZ, capillary budding into FAZ, widening of intercapillary spaces and capillary dropout in perifoveal area.[14]

Clinically Significant Macular Edema (CSME) defined as

1. Thickening of retina within 500µm from centre of macula.

2. Hard exudates with retinal thickening seen within 500µm from centre of macula.[15]

3. A zone of retinal thickening about 1 disc diameter in size, a portion of which is seen within 1 disc diameter from centre of macula.

FFA

Focal edema shows areas of leakage mainly from microaneurysmsin late phases. Diffuse edema shows dilated capillary network with areasof capillary nonperfusion with extravasations of dye in late phases. Ifleakage occurs in flower petal pattern, cystoid macular edema is present

POST OPERATIVE CYSTOID MACULAR EDEMA

It is the most common cause of loss of vision after cataract surgery.A higher incidence is seen when surgery is complicated by vitreous loss, vitreous adhesions to the wound, iris damage or retained lensmaterial. Patients with DM and Uveitis (vasoactive stimuli) are at a greater risk of developing CME

Pathophysiology

- **Vitreous Traction Theory**

Constant constriction and dilatation of pupil created pulling effecton the anterior vitreous strands which was transmitted to the vitreous baseand to

macula by presumed vitreous connections between posteriorhyaloid and surface of macula- VITREOUS TUG SYNDROME.

- **Inflammation Theory**

CME following cataract extraction can be seen in approximately 20% of uncomplicated cases.(6)

Eyes with CME nearly always demonstrate signs of intraocularinflammation and responds to steroid therapy. Aqueous humour contains

biochemically active principles known as Aqueous Biotoxic Complex factorswhich manifest biotoxic effects when it leaves its natural reservoir. Thesediffuse posteriorly through collapsed liquefied vitreous gel. The liquefiedvitreous anterior to retina assumes chemical and osmotic properties thatare not normally present resulting in outpouring of fluid from perimacularcapillaries.Since eye does not contain enzyme 15-Pg dehydrogenase todeactivate prostaglandins, their removal is dependent on active transportpump called Bito'S(23) pump located in the ciliary epithelium which isinoperable for atleast 3weeks post trauma.

- **Anoxic Theory**

An association between CME and systemic conditions likehypertension, diabetes mellitus, arteriosclerotic disease is seen in whichanoxia could be a predisposing factor.

CME In ACIOL

Chronic uveal irritation may either stimulate production of intraocular inflammatory substances or may retard the absorption or removalof these substances by non pigmented epithelium of ciliary body. AnACIOL can press against anterior surface of iris or apply constantpressure over ciliary body which could trigger anterior uvealinflammation.

CME In PCIOL

1. No pupillary distortion and intact posterior capsule

a. Sulcus fixated IOL – chronic irritation of uveal tissuebecause of direct contact can lead to CME

b. In the bag IOL – Incidence of CME is low.

2. Pupillary distortion – Distortion by pupillary capture or bysynechiae between remnants of anterior capsule and posteriorsurface of iris can lead to persistent uveal irritation leading toCME. The degree of irritation is not as great as produced by pupildistorted by wick of vitreous adherent to corneoscleral wound.

CME In Phacoemulsification

There could be a greater incidence during learning curve becauseof increased intraocular manoeuvring and greater risk of complications

CME In Posterior YagCapsulotomy

1. Primary Surgical Posterior Capsulotomy

There is a higher incidence of CME because posterior capsule acts as a barrier to posterior diffusion of inflammatory mediators and to anterior movement of vitreous.

2. Secondary Nd-YagCapsulotomy

- Broken capsule no longer acts as a barrier for posterior

diffusion of inflammatory mediators.

- Inadvertent rupture of anterior hyaloid face.
- Post laser intraocular inflammation.

CME IN UVEITIS

Pathophysiology

Disruption of blood retinal barrier secondary to inflammatorymediators like Cytokines, Interferon-gamma, Interleukin-2 and 10, TNF-alpha .

THEORIES CONCERNING ORIGIN OF CYSTS OF CME

Intracellular Theory

Yanoff et al. proposed that cysts develop from degeneratingMuller's cells. Initially these cells demonstrate edema which graduallyincreases until cytoplasm of cells begin to develop vacuoles. Theseedematous cells gradually expand until cell walls break and adjoiningcells form larger cavities leading to cysts in CME. A breakdown in blood retinal barrier or anoxia is the prime cause for edema.

Extracellular Theory

Gass, Anderson and Davis proposed that cysts arise fromexpansion of extracellular spaces of retina by serous exudation seen within the OPL and inner nuclear layer. This involves leakage of serous exudates from perifovealintraretinal capillaries and sometimes from disc capillaries. These exudates form small cavities with fluid in OPL of Henlewhich acts like a sponge because of peculiar nature of macula.

CLINICAL FEATURES OF CYSTOID MACULAR EDEMA

SYMPTOMS

It is usually asymptomatic. If severe, defective vision,metamorphosia, scotomas can occur.

SIGNS

Fundus

Characteristic honey comb like lesion showing one or more cystoid spaces centrally with many small oval spaces around them.

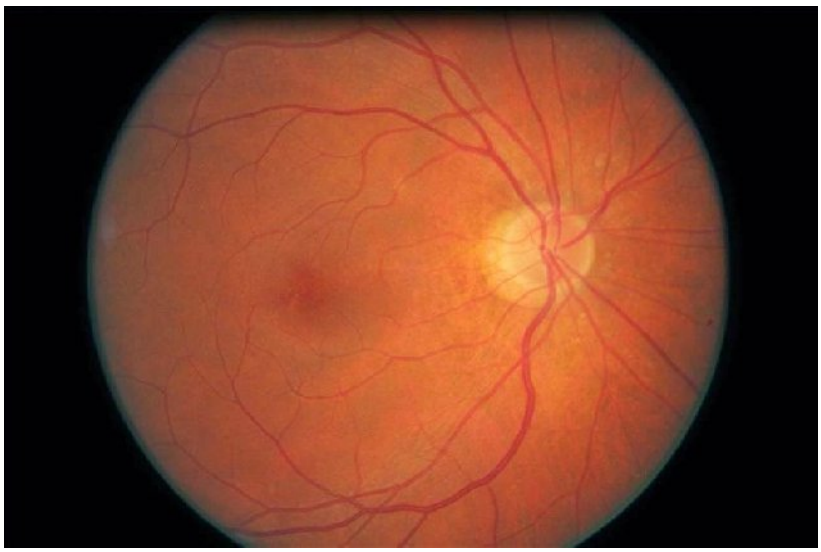
Sometimes only loss of foveal reflex is seen.

Cystoid spaces are best seen with red free filter which makes innerwalls visible.

Retina may be markedly thickened and lesion as large as 1.5 – 2 disc diameter. Some cases are associated with disc edema.

Anterior segment- Usually shows signs of inflammation. Theanterior hyaloid face is broken or intact and vitreous shows cells, vitreousopacities and posterior vitreous detachment.

COLOUR FUNDUS PHOTOGRAPH OF CYSTOID MACULAR EDEMA



CLINICAL EVALUATION OF CYSTOID MACULAR EDEMA

1. VISUAL ACUITY

Best corrected visual acuity and with pin hole should be assessed.Loss of vision depends on the involvement of macula. Severity of visionloss correlates well with amount of macular edema.[14]

2. PERIMETRY

Field charting by perimetry may reveal scotoma corresponding toareas of involvement in fundus.

3. COLOUR VISION

The most common defect is blue . In diabetes the sensitivity of bluecones is depressed. These are best detected by Farnsworth munsell 100 hue test.

4. DIRECT OPHTHALMOSCOPY

Monochromatic light is better for detecting subtle macular changes,hence red free light is used. But lack of stereoscopic view is a disadvantage.

5. SLIT LAMP BIOMICROSCOPY WITH 90D, 78D, HRUBY LENS, GOLDMANN THREE MIRROR CONTACT LENS

The optical section of convex anterior walls of cysts can be seenoverlying empty vesicles tightly packed together with their interfacespresenting a spidery pattern.

With slit beam, it is possible to see a network of interlacing fine refractile lines by retroillumination. Advantages of using stereopsis and slit lamp optics

6. INDIRECT OPHTHALMOSCOPY

This provides an entire view of retina allows examiner to have anunderstanding of the cause in various pathological features of retina.It gives less magnification.

7. RETINAL PHOTOGRAPHY

Photographs are taken with fundus camera and selected films andcolour filters. Red free light is used for detecting subtle changes in ILMand NFL.

8. STEREOPHOTOGRAPHY

Two photographs are taken alongparallel axes on either side ofdilated pupil producing stereoscopic pair. The separation of images canbe adjusted to reproduce the normal depth relationships and to allowdepth measurements.

9. MADDOX ROD TEST

The patient is asked to look at distant light through a Maddox rod.If the red line is continuous and unbroken, macular function is good.

10. AMSLER GRID TEST

Central distortion of the grids or a relative central scotoma

11. ENTOPTIC IMAGERY

The globe is steadily and firmly massaged through the closed lowerlids, with bare lighted bulb of a torch. The entire vascular tree of theretina is

seen on an orange background. Any blanks or scotoma in thecentral area implies macular involvement.[17]

12. POTENTIAL ACUITY METER

It is for differentiating between visual loss from anterior segmentPathology and macular disease. The PAM attaches easily to a standard slitlamp and projects a Snellen's visual acuity chart into eye using an aperture which is 1.5mmdiameter. The patient is tested at different points on thecornea in an attempt to project through the clearer areas in the lens.

13. MACULAR PHOTOSTRESS TEST OR AFTER IMAGE SCOTOMETRY

The patient is asked to look at a flash light held 2cm from the eye for 10 seconds. The time it takes for visual recovery to one line less than thevisual acuity determined prior to this test is measured. The normalrecovery time is 55seconds.[23]Longer recovery time, upto 90-180 seconds

implies macular dysfunction even though the area may appearanatomically normal. The rapidity of recovery relates to the rate of visual

purple regeneration and rapidity of vitamin A transport from RPE tophotoreceptors. Difference between two eyes is also significant.

14. CLINICAL INTERFEROMETERS

In cases of opaque media, beams of coherent light from two pointsources are directed through the clearest area of the lens to retina.Interference fringes on the retina are formed wherever the two beamsoverlap and by varying the width of the inter fringe pattern, the visualacuity can be determined.

15. ELECTROPHYSIOLOGICAL ASSESSMENT

Foveal ERG is a test of the temporal responsiveness of the central100 of the retina and requires integrity of the outer retinal layers,especially Muller's cells. [19]FERG is usually abnormal in 35% of eyes withCME. Pattern ERG reflects the inner retinal layer function. It is usuallyabnormal in 53% of eye with CME. It is the test of the temporalresponsiveness of central 100 of the retina.

16. VISUAL EVOKED POTENTIAL

In macular disease with fluid accumulation, the VEP showsamplitude reduction depending on the decreased visual acuity with nochange in latency.

IMAGING MODALITIES IN CYSTOID MACULAR EDEMA

- **FUNDUS FLUORESCEIN ANGIOGRAPHY**

FFA is mandatory for following purposes:

- Confirm and document macular changes
- Deciding the management

- Follow up.

In CME, within 1-2 minutes of injection, in the arteriovenous phase, early leakage of the dye in the parafoveal area is seen. In someinstances, focal points of leakage with confluence of leaking areas will be noted. [20]A characteristic flower petal appearance is evident in latearteriovenous phase in the parafoveal region. Fovea itself appears dark and does not always show leakage. The late phase shows marked edemawith persistent pooling of dye in cystoid spaces.

The dark septae in the macular area which compartmentalize thepattern are because of the Muller's fibres. The spaces appear tointercommunicate. Usually there is considerable leakage of dye intovitreous and aqueous anteriorly. In some patients with disc edema, there may be leakage of dye into optic nerve and peripapillary retina.

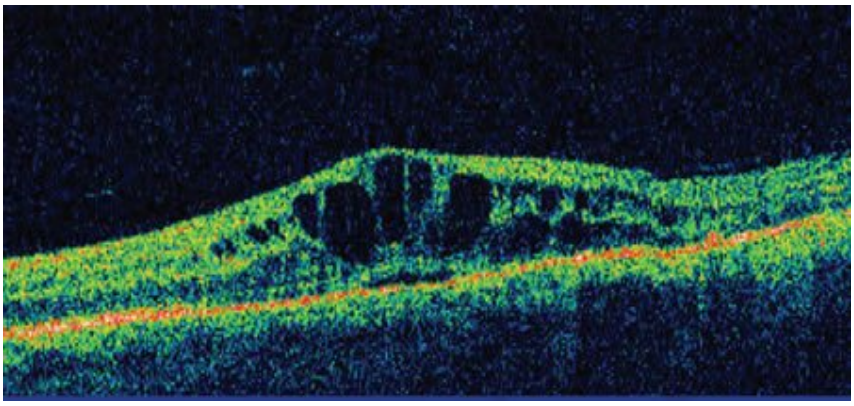
- **OPTICAL COHERENCE TOMOGRAPHY**This investigative modality can also be used for diagnosis andfollow up. It has added advantage of being non-invasive.

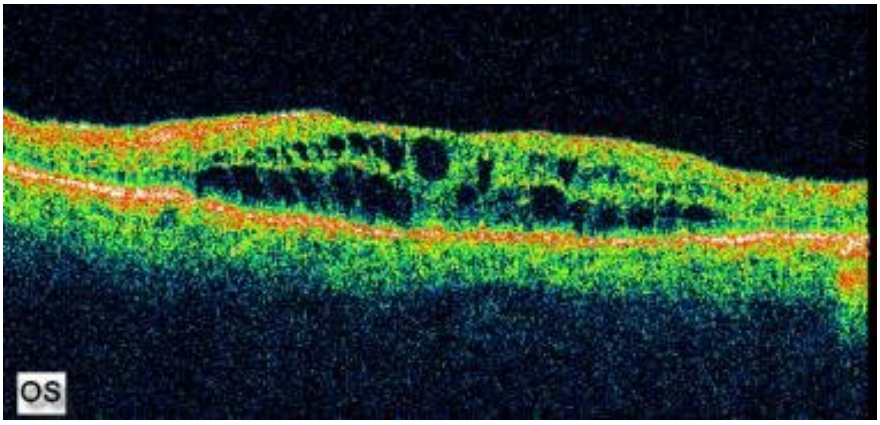
In addition parameters like macular thickness can be quantified onsubsequent visits.

Cystoid spaces confined to outer retina mostly.In long standingcases they fuse to form large cyst.

FFA PICTURE SHOWING CHARACTERISTIC FLOWER PETAL LEAKAGE PATTERN

OCT PICTURE SHOWING CYSTOID SPACES





TREATMENT MODALITIES FOR MACULAR EDEMA

1. LASER PHOTOCOAGULATION

Macular edema secondary to vascular causes like diabeticretinopathy and retinal vein occlusions respond well to focal and gridlaser photocoagulation. Focal laser is used to close microaneurysms andgrid laser stimulates RPE pump to clear the edema.The Early Treatment for Diabetic Retinopathy Study (ETDRS)established guidelines for laser treatment in diabetic macular edema(7).Laser treatment is indicated for

clinically significant macular edema asper definition stated before. Moderate visual loss was reduced to 50% inpatients who received laser therapy in ETDRS.

Grid laser has better response in patients with BRVO but not inCRVO. In Branch Vein Occlusion Study, 65% of grid laser treated eyesgained atleast 2 lines of better vision, compared with 37% of untreated eyes(8).

InCVOS , grid laser did not have any significanteffect on vision, although there was a trend towards better vision inpatients aged <60 years who underwent laser treatment

2. MEDICAL MANAGEMENT

TOPICAL THERAPY

Several studies have documented the utility of topical NSAIDs forboth the prevention and treatment of macular edema after lens removal surgery. A double masked, randomized, placebo controlled trial toevaluate the effect of Ketorolac 0.5% ophthalmic solution on chronicpseudophakic and aphakic CME determined that this drug is beneficial

for the primary outcome measure (Snellen's acuity)(10). The efficacy of topicalFlurbiprofen 0.03% and Indomethacin 1% in preventingpseudophakic CME revealed a reduced incidence of clinical and angiographic CME in the early postoperative period. Recently, two new topical NSAIDs,

Bromfenac 0.09% and Nepafenac 0.1% have been FDA approved for cataract surgery inflammation and pain. [25]There have been reports,particularly for Nepafenac, of their potential for preventing pseudophakicCME.(11) Nepafenac is a prodrug that is converted after cornealpenetration to Amfenac, a potent NSAID. In one animal model,Nepafenac was shown to inhibit prostaglandin in the vitreous humour to a

much greater extent than other traditional NSAIDs. Whether this efficacy will translate into improved prevention or treatment of pseudophakic

CME is currently under investigation.[22]

Corticosteroids drops alone or in combination with NSAID drops,have been studied for the treatment of pseudophakic CME. Treatmentwith topical NSAIDs appears to be more effective than topical steroidsalone. However, combination therapy with topical NSAIDs andPrednisolone acetate was superior in treating macular edema.

Consensus for topical NSAIDs use has not been established formally,

however, the standard of care for many surgeons is to use bothNSAIDs and topical steroids for atleast 1 to 2 days preoperatively and forseveral weeks postoperatively. In higher risk patients, such as those withpre-existing ocular inflammation or diabetes, extended preoperative andpostoperative use is typically employed. Patients must be monitored for

side effects of both NSAIDs (corneal toxicity) and corticosteroid use(increased intraocular pressure).

INTRAVITREAL STEROIDS Corticosteroids by their anti inflammatory effect will contribute to reduction in edema. Increased diffusion by modulation of calcium channels also could account for efficacy of corticosteroids.(15)In last few years, intravitreal triamcinolone (IVTA) has gained

Widespread use as a treatment in all forms of macular edema. IVTAreduces retinal thickening on OCT and improves vision in a substantial

number of patients. Patients with cystoid component respond better. The duration of effect varies and macular edema recurrence and visual decline are observed 4 to 6 months after injection. Repeated therapy is oftenlimited by side effects. Intraocular pressure elevation occurs in about one

third of patients, which can (rarely) require glaucoma surgery.[26]Acceleration of cataract formation, endophthalmitis are othercomplications associated with theprocedure which should be considered and discussed with the patient as a part of informed consent.

IVTA has also been used in macular edema associated with veinocclusion. A recently published 1 year study revealed a short term visual

benefit in patients with CRVO associated macular edema who weretreated for macular edema with IVTA, although their vision generallyreturned to pretreatment levels at 1 year despite repeated injections(16). It

has also been observed that rate of intraocular pressure rise and need for glaucoma surgery appears to be higher in this subset of patients than inthose with diabetic macular edema. The SCORE (Standard Care VsCorticosteroid for Retinal Vein Occlusion) Study, a National Eye Institute

– sponsored randomized clinical trial of intravitreal steroid for BRVO and CRVO associated Macular edema clearly mentions the benefit of IVTAas compared to standard therapy in CRVO. In cases of macular edemadue to BRVO, IVTA and Grid laser shows a comparable response,however, IVTA can be used for macular edema not responding to gridlaser therapy. Finally IVTA has been used in uveitic CME.

INTRAVITREAL DRUG DELIVERY SYSTEMS

Recently multiple alternative steroid delivery devices has arrivedfor treatment of macular edema. Currently four

corticosteroidbasedintravitreal implants under development.Which include:

- Dexamethasone biodegradable implant (Posurdex®)
- Helical triamcinolone acetonide implant (I-vation™)
- Fluocinoloneacetonide implant (Retisert®)
- Fluocinoloneacetonide – based implant that is injectable (Medidur™)

OZURDEX is an intravitreal implant containing 0.7 mg (700 mcg)dexamethasone inthe NOVADUR solid polymer drug deliverysystem.It is effective in reducing macular edema for a period of 3 months.

INTRAVITREAL ANTI VEGF COMPOUNDS

Anti VEGF agents work to restore the normal permeability of theblood retinal barrier. Pegaptanib sodium, an anti VEGF pegylatedaptamer, was used in treating DME .Patients received atleast 3injections at 6 week intervals and were followed for 36 weeks. Thepatients assigned to pegaptanib were more likely to gain atleast 10 letters(34% vs 10%, p=0.003) showed a reduction in centralmacular thickness (42% vs 16%, p=0.02) and at follow up examinationswere deemed less likely to need additional photocoagulation therapy(25% vs 48%, p=0.04).

Ranibizumab (LucentisTM), another intravitreal anti VEGF agentthat has been FDA approved for treatment of wet ARMD was studied in a small series of 10 patients with DME and found to significantly reduce foveal thickness and improve vision(19). Larger randomized trials are necessary to assess the significance of this preliminary finding.[27]In cases of diffuse DME that failed other treatments, intravitreal

injection of Ranibizumab was associated with improved vision and decreased retinal thickness 12 weeks after the first injection(20). Most of the patients received >1 Ranibizumab injection during follow up

Bevacizumab (AvastinTM) is a recombinant, humanized, monoclonal antibody was found to act against VEGF. Avastin has received FDAapproval as an intravenous drug for treatment of metastatic colon cancer.

An intravitreal formulation was first used off label for the treatment of ARMD.

Recent study of intravitrealbevacizumab treatment for macular edema in patients with CRVO revealed a significant decrease in mean central thickness andimproved visual acuity (defined as halving the visual angle) in 14 of 16 eyes(21). Patients had received an average of 2.8 injections and werefollowed for a mean of 3 months. Noadverse outcomes occurred. These encouraging short term results need tobe validated in prospective studies. Bevacizumab has been tried inpatients with macular edema

secondary to uveitis. In one case series, inwhich patients were followed for atleast 2 months after a singleintravitrealinjection, about 70% of treated patients had a decrease infoveal thickness but only 40% of patients had improved visual acuity by>2 lines.(22) Thus for patients with difficult to control macular edema,bevacizumab may be a therapeutic option with reasonably goodoutcomes.

SEQUELAE OF CME

Permanent macular degeneration may arise secondary to prolongedchronic CME. The cystoid spaces of the macula may coalesce together so

that all retinal elements disappear except for the ILM. After the ILM also disintegrates, a lamellar hole is formed which may be one fourth to one third disc diameter in size. In the presence of lamellar hole, the visualacuity may continue to be good because of retention of some percipient

elements.

AIM OF THE STUDY

To study the functional, anatomical and therapeutic outcome of patients with cystoid macular oedema secondary to retinal vein occlusion .

PRIMARY OBJECTIVES

To analyse the prevalence,etiopathogenesis, causes , to analyse the functional and anatomical outcomes (central macular thickness) and current treatment trends.

SECONDARY OBJECTIVES

To analyse most common cause, effectiveness of treatment , frequency of the treatment and follow up of the patients.

INCLUSION CRITERIA

Patients presenting with

1. Patients male or female 18 years old or more.

2. Patients who have not undergone any form of treatment for disease elsewhere.

3. Macular edema secondary to vein occlusion confirmed by fundus evaluation , optical coherence tomography and fluorescein angiography

4. BCVA worser than 6/ 12

EXCLUSION CRITERIA

Patients presenting with

1.Any previous treatment of macular oedema such as intravitrealbevacizumab, laser photocoagulation ,intravitreal triamcinolone or vitrectomy done elsewhere before presenting to our centre.

2. Any significant media opacity which precludes fundus evaluation and renders the diagnosis uncertain.

3. patients with vein occlusion with associated vitreous haemorrhage,macular ischemia at presentation that contributes to decreased visual acuity.

4.Presence of fovealatophy, severe pigmentary changes dense subfoveal haemorrhage , confluent subfoveal hard exudates or any other condition that may influence functional recovery of macular edema**.**

REFRACTORY MACULAR EDEMA

All eyes of retinal vein occlusion induced macular edema received laser photocoagulation and which were refractory to treatment withcentral macular thickness more than 300μm.

All the patients who were pseudophakic received Intravitreal Triamcinolone acetate along with topical NSAIDS when central macular thickness was found to be more than 300 microns.

MATERIALS AND METHODS

This was a prospective study carried out on the patients presenting to uvea and retina services at regional institute was registered, evaluated and followed up during the study period.

About 30 patients were evaluated for a period of 6 months.

A detailed history of the patient will be taken. Complete general examination and vitals measurement will be performed. Ocular examination including visual acuity (usingsnellen's chart with refractive correction), anterior and posterior segment will be evaluated. Slit lamp examination, and fundus examination using Direct, slit lamp biomicroscopy 90D and indirect ophthalmoscopy will be done. Field charting, IOP (goldmannapplanation tonometry) , will be measured. Fundus fluorescein Angiogram and Optical coherence tomography (for assessing the central macular thickness) was done. Laboratory investigations will be analysed. Examination of RS, CVS ,CNS will be performed.

Radiological imaging (carotid Doppler) will be done and analysed for appropriate patients. Patients will also be referred to neurology, physician, diabetologist for expert opinion whenever indicated.

Patients will be managed according to their appropriate diagnosis by either conservative treatment or surgical intervention. Response of the patients to the treatment and the incidence of complications in them will be assessed in the follow-up period.

All patients who had cystoid spaces in macular region on OCT,
who were refractory to treatment like grid and focal laserphotocoagulation in vascular causes and topical anti-inflammatory drugs
and grid laser photocoagulation in pseudophakic cases were subjected to treatment with intravitreal triamcinolone acetonide injection and intravitrealanti VEGF injection. (ranibizumab)

A commercially available Triamcinolone acetonide withoutpreservative (CORTEYE 40mg/ml) is available. Under aseptic techniquesusing 5% povidone iodine and topical antibiotics, 4mg in 0.1ml oftriaminoloneacetonide was injected intravitreally in superotemporalquadrant via parsplana after taking informed consent. After injection, intraocular pressure and central retinal artery perfusion was checked. Patients were instructed to administer topical antibiotics for 1 week.

Similarly commercially available intravitreal anti VEGF Ranibizumab injection (lucentis) was given for patients with cystoid macular edema more than 300 microns. It was mostly was given for phakic eyes. About 0.5mg /0.05 ml was given about 4mm away from the limbus in the supero temporal quadrant under sterile aseptic precautions after taking an informed consent. The intraocular pressure was checked and central retinal artery perfusion was looked for. Patients were followed accordingly.

FOLLOW UP

Patients were followed up 1st week , 3rd week , 6 weeks , 3rd month and as and when required. During there follow up periods at each visit anterior segment examinationby slit lamp biomicroscopy, intraocular pressure with Goldmannapplanation tonometry, visual acuity recording with Snellen's chart andfundus examination by +90 D was done. A repeat OCT was done in the 6th week and 3rd month.

GUIDELINES FOR INTRAVITREAL INJECTION

1. Povidone iodine application for ocular surface, eyelid and eyelashes.

2. Use of eye speculum and avoid contamination of the needle with eyelid margin.

3. Avoid extensive massage of eyelids both pre and post injection.

4. Adequate use of topical anaesthetics.

5. Avoid prophylactic and post injection paracentesis.

6. Intraocular pressure to be checked following injection.

7 . Dilated fundoscopic examination should be done followinginjection to look for central retinal artery perfusion and intraocularlocation of the drug.

MAIN OUTCOME MEASURES

1. Best corrected visual acuity
2. Central macular thickness (μm) with OCT
3. Intraocular pressure (mm Hg) measurement with Goldmann'sapplanation tonometry.

INTRAVITREAL INJECTION

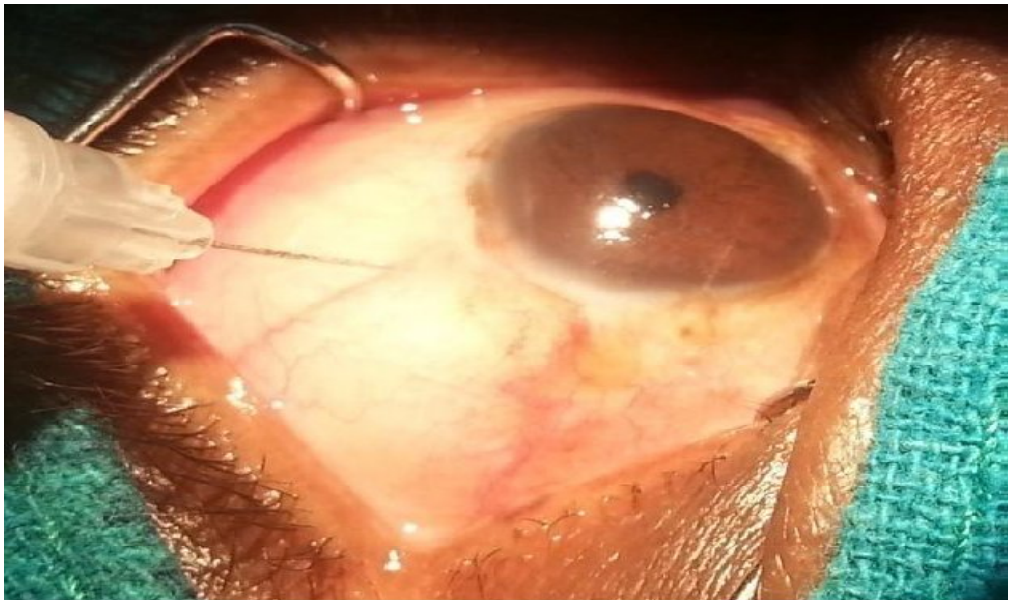


Figure - depict administration of Triamcinolone acetonideintravitreally through parsplana route.

ANALYSIS AND RESULTS

1. AGE DISTRIBUTION

Totalnumber of patients in our study were 30 patients.

The range was between 45 – 65 years including both males and females.

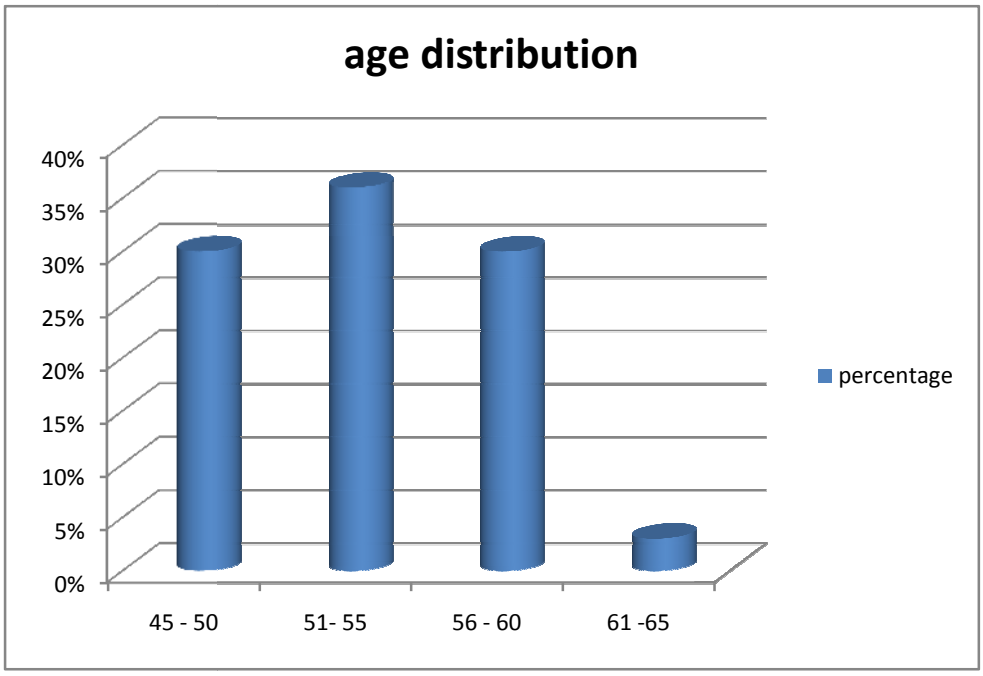
Majority of patients belonged to the age group of 50 to 55 years of age.

An equal distribution was found between the age groups of 45 to 50 and 56 to 60 years.

Mean age of presentation was 53.2 ± 5.6 years. The youngest patient was 46 years old and the oldest patient was 61 years old.

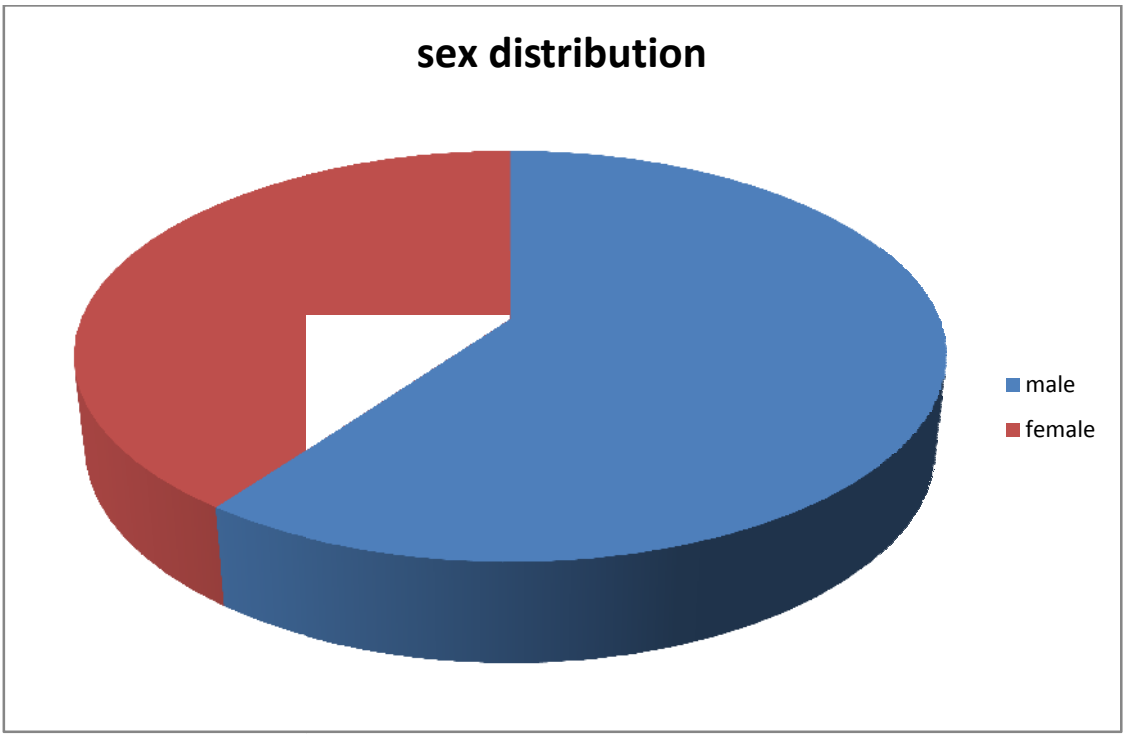
AGE (YEARS)	FREQUENCY	PERCENTAGE (%)

45 – 50	9	30
50 – 55	11	36
55- 60	9	30
60- 65	1	3



2. SEX DISTRIBUTION

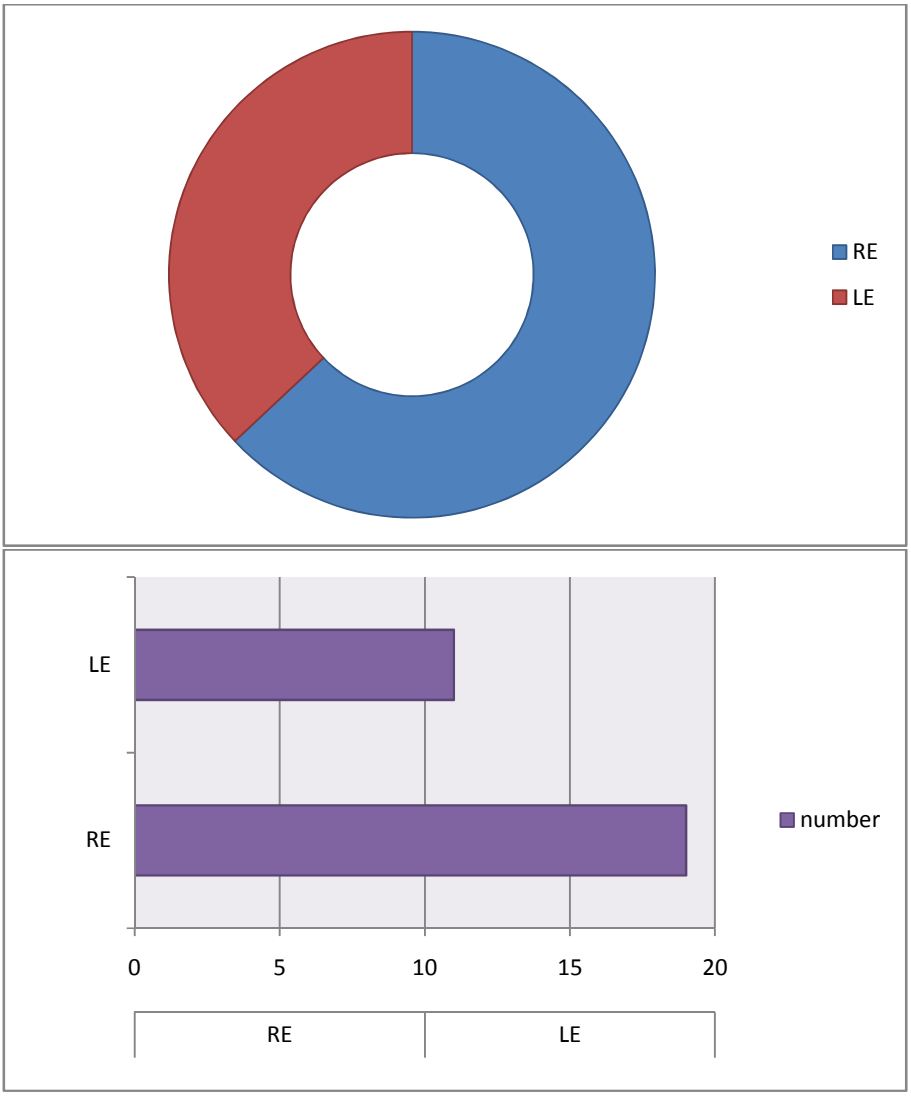
SEX	NUMBER	PERCENTAGE
MALE	18	60%
FEMALE	12	40%



In this study, there was a slight male preponderance, malesaccounting for 60% of patients. Majority of them were in 50 -55 yearsage group.

3.LATERALITY

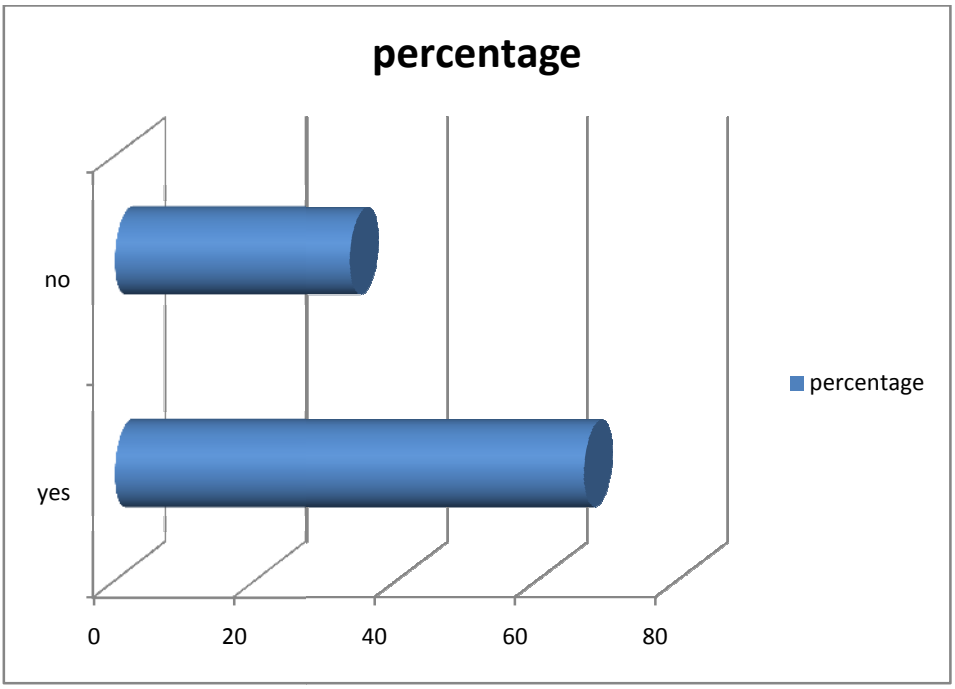
EYE	NUMBER	PERCENTAGE
RIGHT	19	63 %
LEFT	11	37%

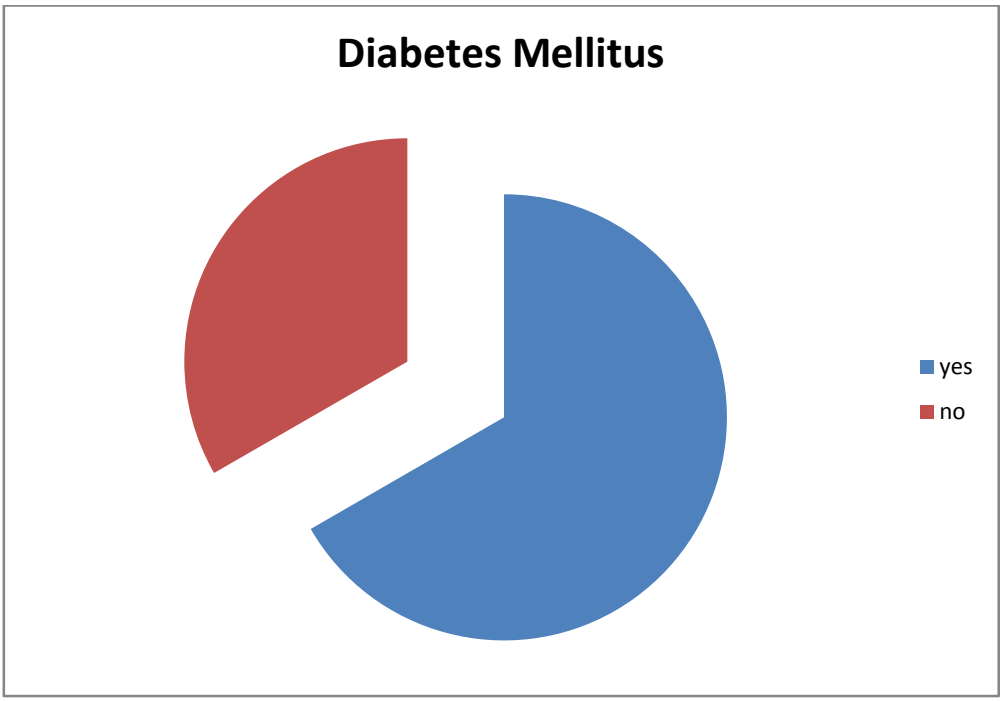


In this study, incidence of cystoid macular edema was more in righteye (63%).

4. **ASSOCIATION OF DIABETES WITH CME**

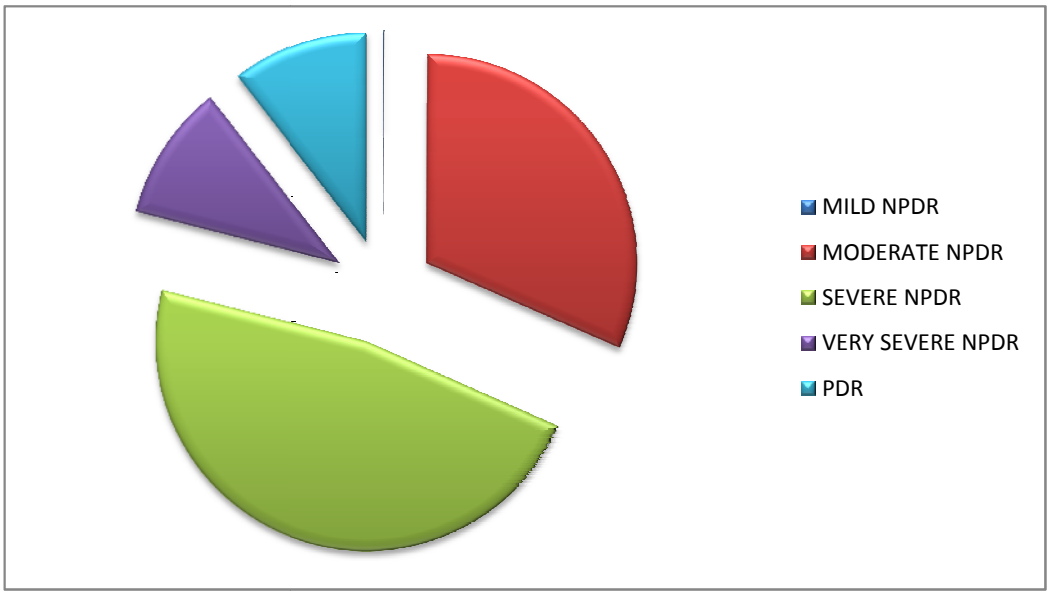
DIABETES	NUMBER	PERCENTAGE
YES	20	66.67%
NO	10	33.33%





In this study it was found that out of the 30 patients evaluated about 20 patients were diabetic. It accounted for about 66.67 % of total number of patients, hence found to be one of the major risk factor.

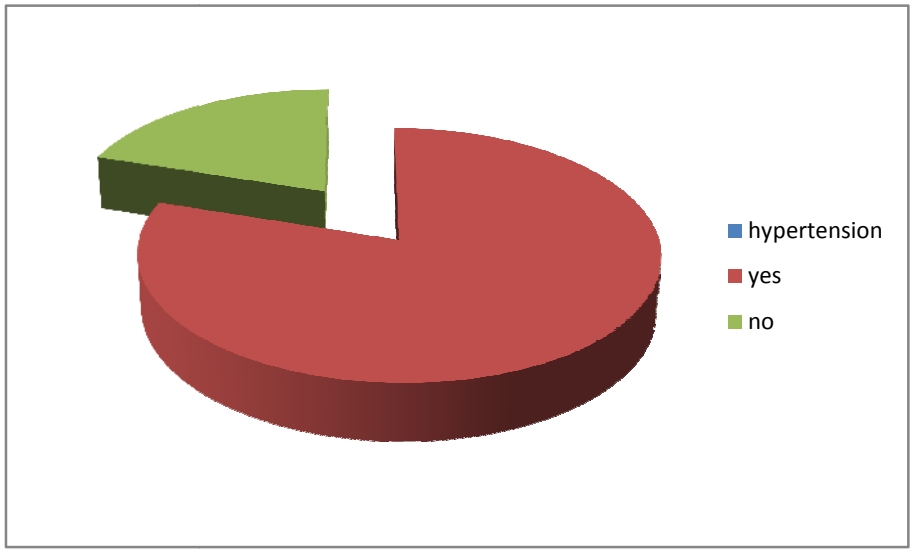
STAGE OF RETINOPATHY	NUMBER	PERCENTAGE
MILD NPDR	1	5%
MODERATE NPDR	6	30%
SEVERE NPDR	9	45%
VERY SEVERE NPDR	2	10%
PDR	2	10%
TOTAL	20	100%



Among the total of 20 diabetic patients MILD NPDR was see in 1 (5%) of the patients, MODERATE NPDR in 6 (30%) patients, SEVERE NPDR in 9 (45 %) patients, VERY SEVERE NPDR in 2 (10%) patients and PDR in 2 (10%) patients

5. ASSOCIATION OF HYPERTENSION.

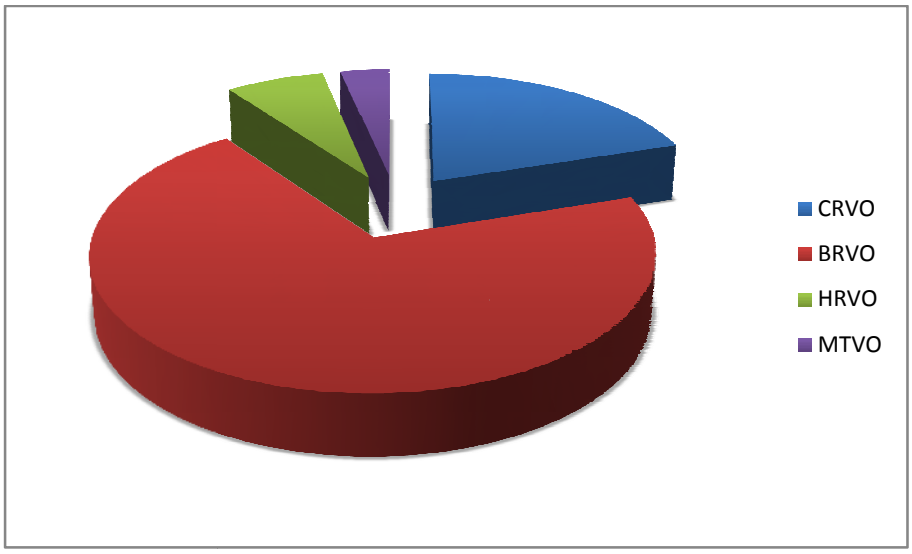
HYPERTENSION	NUMBER	PERCENTAGE
YES	24	80%
NO	6	20%



Among the 30 patients evaluated 26 patients accounting for about 80% were found to be hypertensive. This concludes that hypertension is one of the major risk factor in developing retinal vein occlusion.

6. TYPE OF VEIN OCCLUSION CAUSING CME

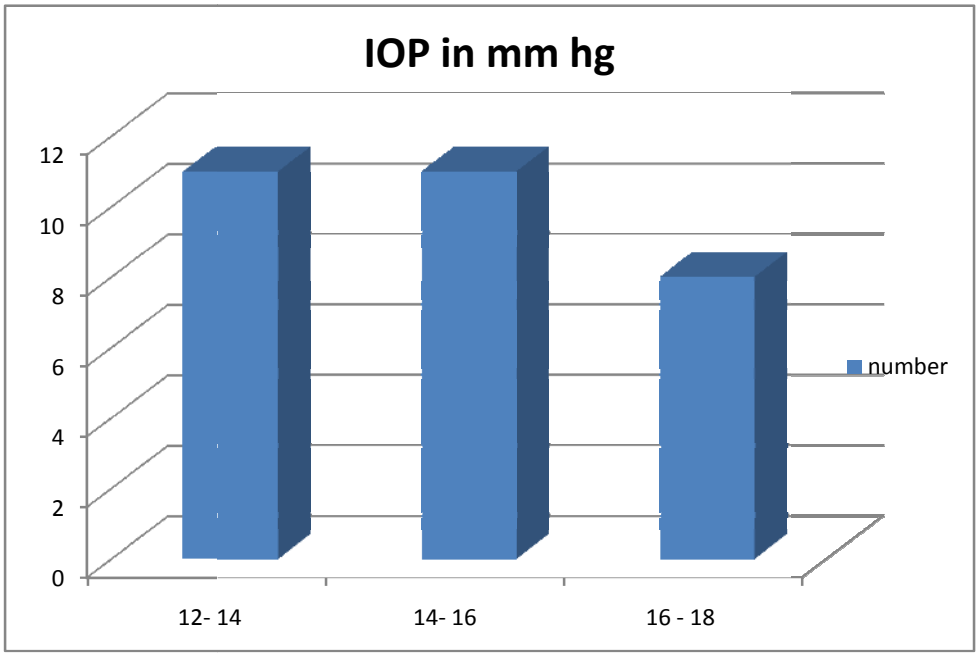
VEIN OCCLUSION	NUMBER	PERCENTAGE(%)
CRVO	6	20
BRVO	21	70
HRVO	2	6.67
MTVO	1	3.37



In this study cystoid macular edema was seen maximum in patients affected with branch retinal vein occlusion. It accounted for total of 70 %. CME due to CRVO was seen in 20% . CME due to hemi retinal vein occlusion was seen in 6.67 %. And CME due to macular tributary vein occlusion was seen in 3.33 % .Hence concluding that the branch retinal vein occlusion is a major cause for developing cystoid macular edema.

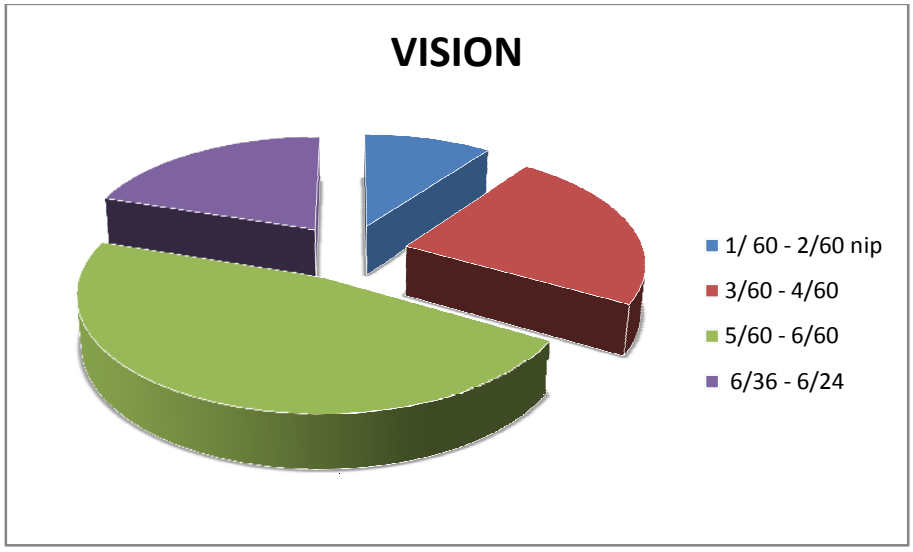
7. RELATIONSHIP OF IOP MEASUREMENTS

The IOP of most of the patients ranged between 12 to 18 mm hg. Most of the patients had a normal IOPof 14 ± 2 mmhg.



8 . VISUAL ACUITY

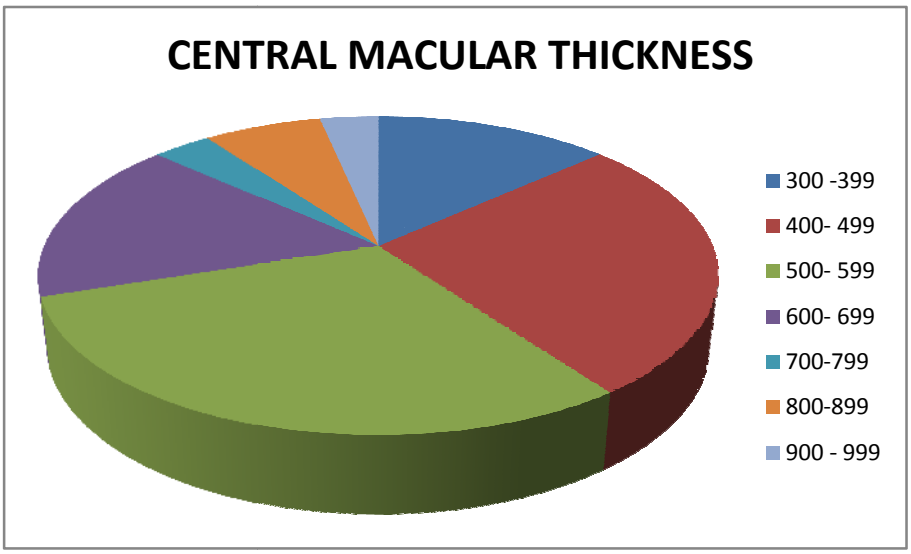
VISION	NUMBER	PERCENTAGE(%)
1/60 – 2/60 NIP	3	10
3/60 – 4/60 NIP	7	23.3
5/60 – 6/60 NIP	14	46.6
6/36 – 6/24	6	20
TOTAL	30	100



In the above study it was found that most of the patients vision ranging between 5/50 – 6/60 NIG NIP .it accounted for 46.6 %. About 10 % of the patients had the vision between 1/60 – 2/60 NIP. 23.3 % of patients had the vision between 3/60 – 4/60 NIP. About 20% patients had a vision between 6/36 – 6/24

9 . CENTRAL MACULAR THICKNESS(CMT) PRETREATMENT

CMT (MICRONS)	EYES AFFECTED	PERCENTAGE (%)
300-399	4	13.33
400 – 499	8	26.67
500 – 599	9	30
600 – 699	5	16.67
700 – 799	1	3.33
800 -899	2	6.67
900 – 999	1	3.33

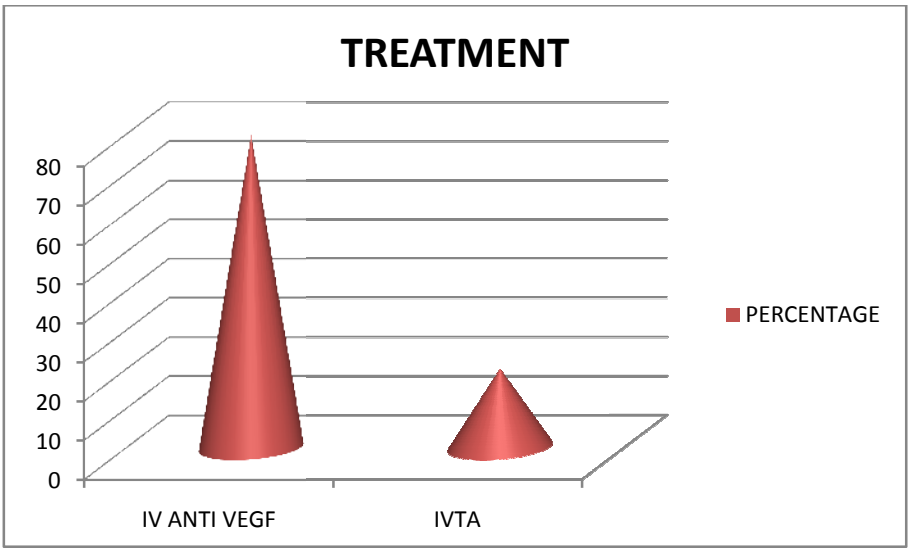


Among the 30 patients evaluated, the highest number of patients had the central macular thickness ranging between 500 – 599µm. The average was found to be 559.6 ± 216.27µmThese patients had a visual acuity ranging between 5/60 to 6/60. Hence all these patients needed surgical intervension.

10 . TREATMENT

All the patients in this study were subjected to surgical intervension based on the OCT finding of central macular thickness of more than 300 microns. These patients had features of cystoid macular edema on FFA as well. About 6 patients among the 30 were found to be pseudophakic. These patients were given intra vitreal triamcinolone acetate. And other patients were give intra vitreal anti VEGF ranibizumab injection.

TREATMENT	NUMBER OF EYES	PERCENTAGE (%)
IV ANTI VEGF (ranibizumab)	24	80
IV TA	6	20



DATA ANALYSIS IN RETINAL VEIN OCCLUSION MACULAR EDEMA

PRE TREATMENT MACULAR THICKNESS

CMT (MICRONS)	EYES AFFECTED	PERCENTAGE (%)
300-399	4	13.33
400 – 499	8	26.67
500 – 599	9	30
600 – 699	5	16.67
700 – 799	1	3.33
800 -899	2	6.67
900 – 999	1	3.33

The average was found to be 559.6 ± 216.27μm

POST TREATMENT MACULAR THICKNESS

CMT	**6 WEEKS**		**3 MONTHS**	
	Numberof EYES	**%**	**Number of eyes**	**%**
< 200	**0**	**0**	**2**	**6.67**
200 – 299	**6**	**20**	**5**	**16.67**
300 – 399	**6**	**20**	**16**	**53.33**
400 – 499	**10**	**33.33**	**6**	**20**
500 – 599	**5**	**16.67**	**1**	**3.33**
600 – 699	**2**	**6.67**	**0**	**0**
700- 799	**1**	**3.33**	**0**	**0**

The mean macular thickness at 6 weeks and 3 months post intervention were 429.5 ± 90.31 μm (S.E – 19.02) and 333.86 ± 58.41 μm (S.E – 11.92) respectively

Paired t test showed that there is a significantdifference in centralmacular thickness before and after Intra vitreal injection at 6 weeks & 3months ' p' values being 0.0179, 0.009 respectively.

The mean reduction in macular thickness at 6 weeks and 3 months post intervention were 276.91 ± 89.53 μm (S.E – 26.99) and 450.55 ±126.40 μm (S.E – 38.11) respectively.

DISCUSSION

AGE DISTRIBUTION - In this study of patients, majority of the patients (75%) belong to 5th and 6th decades. This may be due to the fact that most of cataract surgeries take place in this age group and prevalence of systemic diseases like DM and HT is more common in this age group. We had 9 casesbelow 50 years accounting for 30%of patients. Previous studiesshowed similar involvement. (Daniel M.Taylor et al.(21) Survey ophthalmology)

SEX DISTRIBUTION The study revealed a slight Male preponderance with a male to female ratio of 1.9:1.

LATERALITY

In this study, RE (63%) was more commonly involved than LE(37%),

ETIOLOGY

BRVO shares the highest proportion of 70% followed byCRVO (20%), followed by HRVO (6.6%) last being macular tributary vein occlusion (3.3.%).

CME IN RELATION TO SURGICAL COMPLICATONS

About 2 cases in the above study developed complications like Epiretinal membrane. It was found in patients who presented with CRVO and BRVO respectively with a raise in the macula thickness of more than 3 fold.

CME IN ASSOCIATION WITH VEIN OCCLUSION AND DIABETES MELLITUS

Out of 30 eyes studied, 20 eyes suffered diabetic retinopathy ofwhich 9 patients belonged to severe NPDR stage followed in order bymoderate NPDR(6), very severe NPDR(2), PDR(2), mild NPDR(1)

INTRA VITREAL INJECTIONS AND MACULAR THICKNESS

In a country like India with high number of diabetics, visualmorbidity due to DME is very high. Intravitreal triamcinolone acetonideis a promising therapeutic method for diabetic macular edema. As ofcurrent practice, according to ETDRS, laser photocoagulation isadvocated for DME. However refractory edema can be treated withintravitreal Triamcinolone. Studies focused on macular edema that failedto respond to conventional laser photocoagulation (Martidis(25) et al.,Karacorlu(26) et al., Audren(27) et al., Gilles(28) et al., ophthalmology 2006)stated that IVTA is effective in improving vision, reducing maculararthickness and inducing reabsorption of hard exudates in diffuse diabeticmacular edema.

In cases of macular edema due to venous occlusions, the recentlyconcluded SCORE(31) study clearly mentions the benefit of IVTA ascompared to standard therapy in CRVO. In BRVO, Triamcinolone

andGrid laser shows a comparable response, however triamcinolone can beused for macular edema not responding to laser therapy.IntravitrealAnti VEGF also showed a reasonably good results.

There was a significant reduction in central macular thickness in all patients both at 6 weeks and3 months follow up period.Maximum reduction in macular thickness at 6 weeks and 3 months was observed in patients who had developed cystoid macular edema due to branch vein occlusion.

VISUAL ACUITY

All the patients treated with the intra vitreal injections showed a significant improvement in the visual acuity after a follow up period of 3 months. In my study patient presenting with a vision of 5/60 improved to 6/12 by 3 months after the treatment with IVTA. Similarly patient presenting with a vision of 6/36 NIG improved to vision of 6/9 after treatment with intravitreal Anti VEGF. Here in my study IVTA was equally effective in the treatment of CME and was found safe in patients with pseudophakia.

INTRAOCULAR PRESSURE

Remained within normal limits all through the follow up periods.

CATARACT

A recent study by Gillies(28) et al., has demonstrated that steroidrelated cataracts are more likely to form in patients who are steroidresponders. In this study , as the follow up period was only 6months,and lens changes were not excluded in the beginning, progression of lenschanges could not be assessed.

RECURRENCE OF MACULAR EDEMA

Massin(27) et al., looked at patients unresponsive to laserphotocoagulation and found a significant difference between CMT ofeyes injected with 4mg IVTA and control eyes but effect was no longersignificant at 24 weeks because of recurrence of macular edema. Thistransient reduction in CMT correlated also to visual acuity.

In this study, about 50% eyes needed a repeat injectionwho had a follow up of over 6 months due to weaning effect of the drug.

COMPLICATIONS

Refractory cases who did not show visual acuity improvementdespite reduction in macular thickness had complications like ERM accounting for 6.67%

SUMMARY

This is a one year prospective ,non randomized clinicalTo compare the functional , anatomical and therapeutic outcome of patients with cystoid macular oedema due to retinal vein occlusion .

The main aim was to evaluate the improvement in visual acuity anddecrease in macular thickness caused due to retinal vein occlusions.

There was no increase in the inta ocular pressure.In this study, 30 patients were analysed and followed upfor a period of 6 months.

The mean age of presentation is 53.2 ± 5.6 years. The youngest patient was 46 years old and the oldest patient was 61 years old.

A slight male preponderance with male : female ratio of 1.9 :1

BRVO shares the highest proportion of 70% followed by CRVO (20%), followed by HRVO (6.6%) last being macular tributary vein occlusion (3.3.%).

Out of 30 patients studied, 20 eyes suffered diabetic retinopathy, ofwhich severe NPDR outnumbered other stages.

MACULAR THICKNESS

Based on analysis of data available, there has been a substantialreduction in central macular thickness after IVTA andIntravitreal anti VEGF administration during 6^{th} week and 3^{rd} month follow up.

VISUAL ACUITY

All the patients treated with the intra vitreal injections showed a significant improvement in the visual acuity after a follow up period of 3 months. In my study patient presenting with a vision of 5/60 improved to 6/12 by 3 months after the treatment with IVTA. Similarly patient presenting with a vision of 6/36 NIG improved to vision of 6/9 after treatment with intravitreal Anti VEGF

LIMITATIONS OF STUDY

Lens changes (status) becomes a major confounding factor while analysing the complications of intravitreal Triamcinolone. This has not been addressed in the study.

While considering the weak correlation between reduction inmacular thickness and visual acuity improvement, the possibilityofmacular ischemia has to be ruled out which the study did not take intoaccount.

Larger study group necessary to quantify the actual magnitude ofbenefit of this treatment modality with comparison to other therapies.Longer follow up is necessary to document the post injectionrecurrences and progression of cataract.

CONCLUSION

Cystoid macular edema presented mostly in the 5th and 6th decadewith slight male preponderance.This study describes supero temporal BRVO as the leading etiology.

This study describes diabetic retinopathy as associated etiology with highest association with severe NPDR stage

Among the 30 patients evaluated 26 patients accounting for about 80% were found to be hypertensive. This concludes that hypertension is one of the major risk factor in developing retinal vein occlusion.

The fall in Mean macular thickness after IVTA ansd anti VEGF injections was significant in all patients analysed.Etiologywise ,BRVO patients seem to be benefitted more with greater reduction in mean macular thickness.Among these patients visual acuity improvement was on anaverage 2 Snellen line post the treatment.

Intravitreal Triamcinolone acetonide is a promising therapy inrefractory cystoid macular edema with established safety andeffectiveness.

Further study with a longer follow-up period and largerseries is warranted to assess the treatment's long term efficacy and safetyand the need for retreatment.

COMPARISON BETWEEN PRE AND POST TREATMENT MACULAR OCT

CASE 1

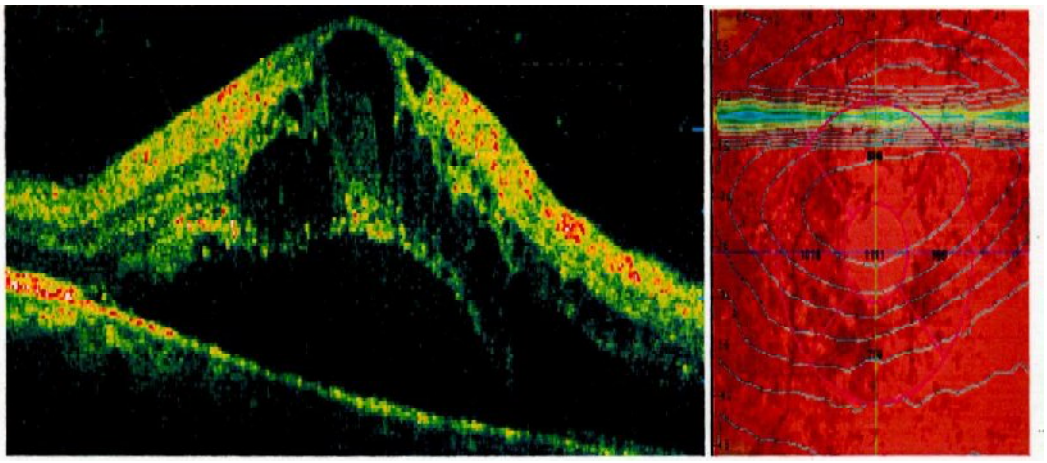


FIGURE 1: Pre injection: OCT shows cystoid spaces intraretinally with serous macular detachment. CMT 903 µm

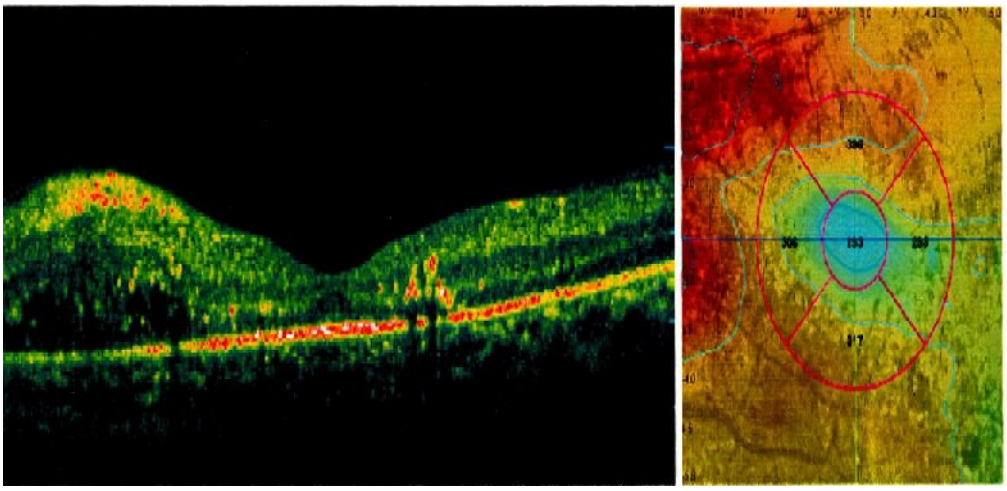


FIGURE 2: 3 monthsPost injection: Repeat OCT shows reduction in cystoid spaces with macular thickness 190 µm

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CASE – 2

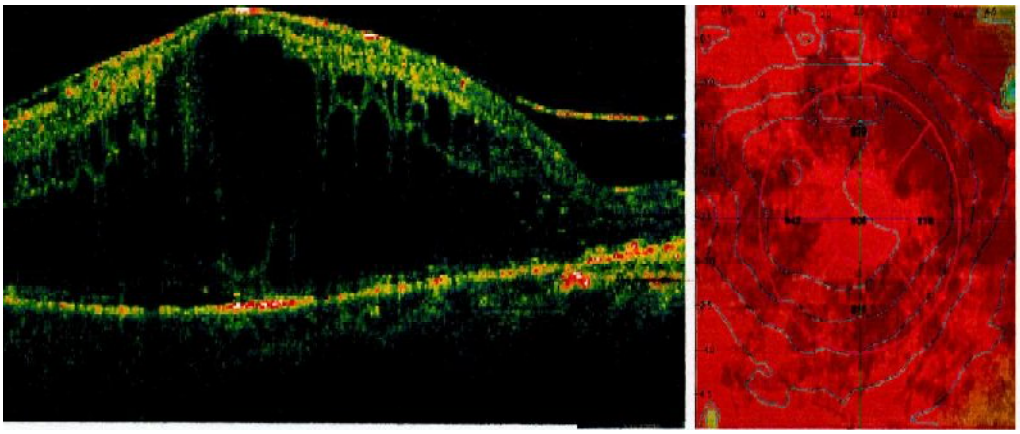

FIGURE 1: Pretreatment

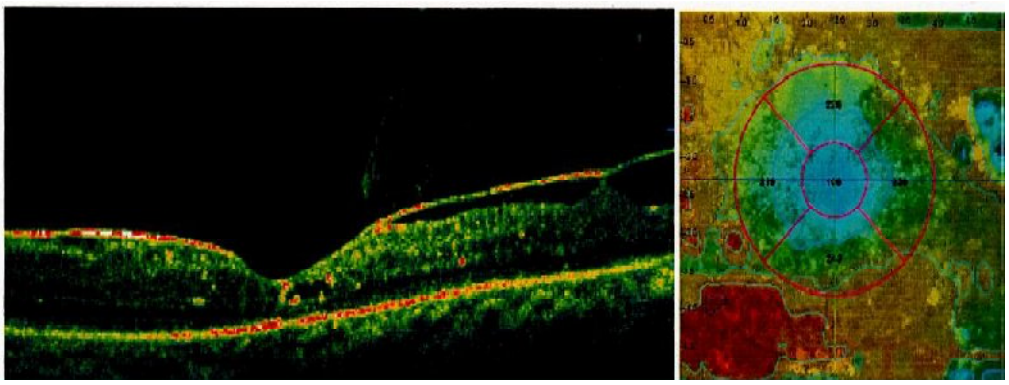

FIGURE 2: Post treatment 3 months showing a reduction in CMT.

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PROFORMA:

NAME :

AGE/SEX :

ADDRESS:

I.P NO :

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

H/O defective vision / field of vision

H/O micropsia, macropsia.

H/O diabetes, hypertension, chronic illness

PAST HOSTORY:

H/O similar episodes in the past

H/O diabetes ,hyperlipidemia, stroke

H/O renal disease

H/O oral contraceptive pills.

H/O any previous treatment , previous surgery (cataract , ppv)

H/O recurrent episodes of redness , watering

H/O glaucoma

FAMILYHISTORY

H/O diabetes

H/O hypertension,

TREATMENT HISTORY

Any medical/surgical treatment underwent earlier

GENERAL EXAMINATION

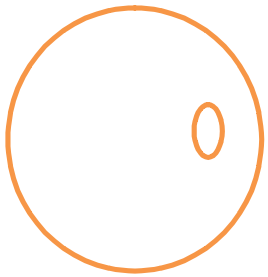
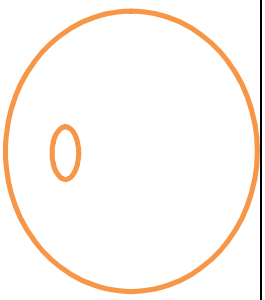
Built

Nourishment

Anaemia/jaundice/cyanosis/clubbing/lymphadenopathy

Vitals-pulse, temperature, blood pressure, respiratory rate

OCULAR EXAMINATION

RIGHT EYE	EXAMINATION	LEFT EYE
	Visual acuity	
	Eyelids	
	Extraocular movements	
	Conjunctiva	
	Cornea	
	Anterior chamber	
	Iris	
	Pupil	
	Lens	
	Fundus examination	
	Intraocular pressure	

OTHER SYSTEMS

CNS,CVS

OTHER CONSULTATION

Neurology/diabetology/physician/hematology/rheumatology opinion

PROVISIONAL DIAGNOSIS

INVESTIGATIONS

Complete hemogram with ESR (in young patients work up for thrombophilia, auto antibodies, and homocystiene)

FBS, Fasting lipids

Urine albumin and sugars

ECG , ECHO

FFA

OCT

FINAL DIAGNOSIS

TREATMENT

FOLLOWUP PERIOD

I week

III week

VI week

3 months

AS AND WHEN NEEDED

INDEX TO MASTER CHART

SEX - M – Male

F – Female

DM - Diabetes mellitus

Y – Yes

N – No

1– Mild NPDR

2 – Moderate NPDR

3– Severe NPDR

4– Very severe NPDR

5 – PDR

HT - Hypertension

Y – Yes

N – No

EYE AFFECTED - RE – Right eye

LE – Left eye

DIAGNOSIS –

1 –Central retinal vein occlusion with cystoid macular edema

2 – Branch Retinal vein occlusion with CME

3 – Hemi Retinal vein occlusion with CME

4 – Macular Tributary vein occlusion

VA – Visual acuity

PH – Pin hole

NIP – Not improving with pinhole

IOP – Intraocular pressure (in mmHg)

OCT – Optical coherence tomography

CMT – Central macular thickness (in μm)

CPL – complications

ERM – Epi retinal membrane

TREATMENT

V – intravitreal anti VEGF injections (ranibizumab)

T – intravitreal triamcinolone injection

NAME	AGE	SEX	COMPLAINTS	DM	HTN	EYE AFFECTED	DIAGNOSIS	VN ON PRESENTAION	CMT	IOP(mmhg)	Treatment	VN 3WEEK	VN 6 WEEK	VN 3 MONTH	CMT 6 WK	CMT 3MT	CMPL
SHANKAR	48	M	DV, F,	y3	Y	RE	2	2/60 NIG NIP	664	16	V	2/60 NIG NIP	2/60 NIG NIP	3/60NIG NIP	540	480	-
KARUPAYI	54	F	DV, F, M	Y4	Y	RE	2	6/36 NIG NIP	345	14	T	6/36 NIG NIP	6/36 NIG NIP	6/36 NIP	280	280	-
FATIMA	55	F	DV,FL,	Y3	Y	RE	2	6/36 NIG NIP	380	16	V	6/36 NIG NIP	6/18 NIG NIP	6/9 NIP	234	210	-
VIJAYA	53	F	DV, F,	y1	Y	RE	2	3/6 0NIG NIP	550	14	V	3/60 NIG NIP	4/60 NIG NIP	4/60 NIP NIG	446	390	-
RANGANTHAN	47	M	DV, M,F	Y5	Y	RE	1	1/60 NIG NIP	903	14	V	1/60 NIG NIP	1/60 NIG NIP	2/60 NIG NIP	780	540	ERM
ARUMUGAM	45	M	DV, F	Y5	N	RE	2	5/60 NIG NIP	448	16	T	5/60 NIG	6/60 NIG	6/60 NIP	347	304	-
VIJAY KUMAR	54	M	DV, F	Y4	Y	RE	2	6/24 NIG NIP	307	18	V	6/24 NIG NIP	6/18 NIG NIP	6/12 NIP	230	190	-
ELLAPPA	58	M	DV, F	Y3	N	LE	2	5/60 NIG NIP	448	14	V	5/60 NIG	5/60 NiG	6/60 NIP	380	310	-
MALLA REDDY	59	M	DV, M	N	N	RE	1	1/60 NIG NIP	846	16	V	3/60 NIG NIP	3/60 NIG NIP	4/60 NIP NIG	640	420	-
POONGODAI	51	F	DV, M	y2	Y	RE	2	5/60 NIG NIP	448	18	V	6/36NIG NIP	6/18 NIG NIP	6/12 NIP	238	238	-
MARY	49	F	DV, FL	Y2	Y	RE	1	2/60 NIG NIP	776	14	V	3/60 NIG NIP	3/60 NIG NIP	4/60 NIP NIG	550	420	-
RAJESH	48	M	DV, FL	Y3	Y	LE	2	3/60 NIG NIP	689	16	V	3/60 NIG NIP	3/60 NIG NIP	4/60 NIP NIG	546	434	-
KAVIN RAJ	55	M	DV, FL	N	Y	LE	2	3/60 NIG NIP	654	18	V	4/60 NIG NIP	5/60 ph 6/60	6/36NIP	442	310	-
MAGESAN	52	M	DV, M,F	Y2	Y	LE	2	4/60 NIG NIP	567	12	T	5/60 NIG	5/60 NiG	6/60 NIG NIP	452	380	-
SEKAR	58	M	DV, M	Y2	Y	RE	2	5/60 PH 6/60	456	18	V	6/24 NIG NIP	6/18 NIG NIP	6/18 ph 6/9	231	190	-
RAJA	54	M	DV, F	N	Y	RE	2	6/60 NIG NIP	434	16	T	6/36 NIG NIP	6/36 NIG NIP	6/36 NIG NIP	338	304	-
MUNNIYAMMAL	61	F	DV, FL , M	Y3	Y	LE	3	4/60 NIG NIP	565	14	V	5/60 ph 6/60	6/60 NIG	6/36NIP	436	330	-
SELVI	48	F	DV , F,	N	Y	LE	2	5/60 NIG NIP	578	16	V	5/60 NIG	6/60 NIG	6/36 NIG NIP	446	390	-
KASTURI	49	F	DV, F	Y3	N	LE	4	6/36 NIG NIP	345	16	T	6/24 NIG NIP	6/24 ph 6/12	6/12 NIP	290	234	-
REVATHI	57	F	DV, F , M	N	N	RE	2	6/60 NIG NIP	567	18	V	6/36 NIG NIP	6/36 NIG NIP	6/24NIG NIP	430	330	-
RAVIKUMARAN	54	M	DV, F	Y2	Y	RE	1	5/60 PH 6/60	445	14	T	6/36 NIG NIP	6/36 NIG NIP	6/24 NIG NIP	326	304	-
MAHALAKSHMI	46	F	DV, F	Y3	Y	RE	2	2/60 NIG NIP	887	12	V	2/60 NIG NIP	2/60 NIG NIP	4/60 NIP NIG	645	430	ERM
SELVARAJ	55	M	DV, M	N	Y	LE	2	5/60 PH 6/60	567	14	V	6/60 NIG NIP	6/60 NIG	6/36NI P	454	348	-
MOHAN KUMAR	56	M	DV, M	y3	Y	LE	1	5/60 NIG NIP	590	16	V	6/36 NIG NIP	6/36 NIG NIP	6/24 NIG NIP	490	330	-
DELLI BABU	52	M	DV, FL, M	N	Y	LE	2	6/60 NIG NIP	446	18	T	6/60 NIG NIP	6/60 NIG	6/36 NIG NIP	345	310	-
POONGAVANAM	58	F	DV, FL	N	Y	RE	2	4/60 NIG NIP	678	16	V	6/60 NIG NIP	6/60 NIG	6/36 NIG NIP	565	310	-
RAJALAKSHMI	47	F	DV, M	y 3	Y	LE	1	5/60NIG NIP	604	18	V	5/60 NIG	5/60 NiG	6/60 NIG NIP	540	420	-
NAGAMMAL	58	F	DV, FL. M	N	Y	RE	3	6/ 60 NIG NIP	578	16	V	6/60 NIG NIP	6/60 NIG	6/36 NIP	468	310	-
DURAI PANDI	59	M	DV, F	Y3	Y	RE	2	6/60 ph 6/36	567	18	V	6/60 NIG NIP	6/60 NIG	6/36 NIG NIP	446	360	-
CHINNA THAMBI	56	M	DV, F, L	N	Y	RE	2	6/60 NIG NIP	456	14	V	6/36 ph 6/18	6/18 NIG NIP	6/12NIG NIP	330	210	-